REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME IV.

Session of 1903–1904.

EDITOR:

GEORGE CARPENTER, M.D.

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10/1/08.

Hon. Secretaries:

London { GEORGE A. SUTHERLAND, M.D.,
EDMUND CAUTLEY, M.D.,

Provincial R. H. ANGLIN WHITELOCKE, C.M. (Oxford).

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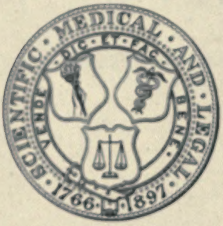

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OFFICE BEARERS AND MEMBERS OF THE COUNCIL FOR THE SESSION OF 1904-1905.

ELECTED AT THE ANNUAL GENERAL MEETING.

Council (1904-1905).

Those marked thus (*) served on the Council or held office during last session, 1903–4.

*J. FORD ANDERSON, M.D.
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*H. R. HUTTON, M.B. (Manchester).
*PERCY LEWIS, M.D. (Folkestone).
W. A. MACKINTOSH, M.B. (Stirling).
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Honorary Treasurer.

*R. CLEMENT LUCAS, B.S.

Honorary Editor of Reports.

*GEORGE CARPENTER, M.D.

Honorary Secretaries.

London { *GEORGE A. SUTHERLAND, M.D.
EDMUND CAUTLEY, M.D.

Provincial *R. H. A. WHITELOCKE, C.M. (Oxford).

LIST OF OFFICE-BEARERS SINCE THE FORMATION OF THE SOCIETY IN THE YEAR 1900-1901.

Dates.	Chairmen of Council.	Honorary Secretaries.		Honorary Treasurer.	Editor of the 'Reports.'
		London.	Provincial.		
1900–1	A. E. Sansom, M.D.	Sydney Stephenson, C.M.	Charles H. Willey, C.M. (Sheffield).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1901–2	A. E. Sansom, M.D.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1902–3	A. H. Tubby, M.S.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1903–4	Sydney Stephenson, C.M.	George Carpenter, M.D. G. A. Sutherland, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.

RULES.

Name.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

Object of the Society.

2. The object of the Society shall be the study of disease and of allied subjects in children.

Constitution.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

Qualification for Membership.

4. All duly registered medical men shall be eligible for membership.

Officers.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, an Honorary Editor of 'Reports,' Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council and manage the affairs of the Society.

Ordinary Members.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

Form of Admission.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

Temporary Members.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

Honorary Members.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

Resignation and Expulsion of Members.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially

for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

Subscriptions.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

Election of Officers.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer, may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

Honorary Treasurer.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and business-like way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one of the Honorary Secretaries. He shall prepare an annual statement of accounts, which shall be audited by two ordinary

members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

Honorary Editor of 'Reports.'

13A. The Honorary Editor of the 'Reports' shall edit the Society's 'Reports' and he shall keep all papers and communications and illustrations intended for publication in the 'Reports.' He shall be *ex-officio* a member of the Council.

Honorary Secretaries.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society, and, unless otherwise decided by the Council, shall act as editors of all publications authorised by the Council. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

Meetings.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of

particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

Chairman.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

Publications.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

Visitors.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take

part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

Council.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded. The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. It shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

REPORT OF THE COUNCIL, 1903–4.

Read at the Annual General Meeting, held at the Hotel Cecil, on Friday, July the 15th, 1904.

The Council have pleasure in reporting that the Society, which started with an original membership of 102 in 1900, now numbers 304 members.

The first meeting of the session was held at the Hotel Cecil on Friday, October the 16th, 1903, and was followed by the Annual Dinner, fifty-five members and their guests being present, Dr. A. E. Sanson in the chair.

The Provincial Meeting was held at Bristol on Saturday, June the 18th, cases and morbid specimens of great interest being shown at the Hospital for Sick Children, St. Michael's Mount, previous to the Meeting by Drs. Bertram H. Rogers, E. Cecil Williams, Fortescue-Brickdale, and Theodore Fisher, and by Messrs. Charles A. Morton (introduced), H. W. Kendall (introduced), and H. Elwin Harris (introduced). After tea an adjournment was made to the Medical Library, where the meeting took place under the Presidency of Dr. Theodore Fisher. Cases and microscopical specimens were shown and papers read by Dr. J. Michell Clarke (introduced) and Dr. Bertram Rogers, and by Messrs. Paul Bush, C.M.G. (introduced), J. Lacy Firth (introduced), R. G. Poole Lansdown (introduced), D. S. Gerrish (introduced), and Charles A. Morton (introduced). The meeting was well attended, some fifty members and visitors being present.

The Provincial Dinner was held subsequently at the Clifton Down Hotel, Clifton, Dr. Frederick Taylor in the chair.

The meetings of the Society continue to be well attended, the average number of signatures in the attendance book being thirty-five. Ten meetings were held during the Session, one of them being an extra meeting in December, 1903, the delivery of the Wightman Lecture having been unavoidably postponed in May, for which date it had been announced, until the Annual General Meeting; and the omission was supplied by the reading of a paper and a lantern demonstration by Mr. Harold Stiles, of Edinburgh, on "Delayed Chloroform Poisoning."

The Council have appointed Dr. Henry Ashby, of Manchester, to deliver the Wightman Lecture during the Session 1904–5.

The Hon. Treasurer's Balance-sheet shows that the Society is in a sound condition financially.

TREASURER'S STATEMENT, 1903–1904.

Receipts.	£	s.	d.
Balance brought forward	263	16	4
Subscriptions	346	7	0
Dividends on Consols	4	1	10
	£614	5	2

Payments.	£	s.	d.
G. Pulman and Sons, for Printing	11	19	8
„ „ „	2	10	0
Adlard and Son, for 'Reports'	164	16	11
„ for Printing, etc.	40	3	6
Medical Society	16	16	0
Dr. Fisher	1	9	0
Dr. Sutherland	3	2	0
Dr. A. E. Jones	1	15	0
Dr. George Carpenter	2	0	0
Mr. H. Dickinson	16	18	3
Treasurer's Petty Cash	3	0	0
Cheque Book	0	5	0
Purchase of £228 13s. 6d., 2½ per cent. Consols @ 87⅞	200	0	0
	464	15	4
Balance carried forward	149	9	10
	£614	5	2

INVESTMENTS.

Consols, 2½ per cent. £228 13 6

WIGHTMAN LECTURE FUND.

Balance brought forward £20 0 0

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct.

The Auditors desire to draw the attention of the Society to the fact that the cost of Printing, exclusive of the Annual Reports, has increased from £23 3s. 7d. in the Session 1902–3 to £54 13s. 2d. in the Session 1903–4.

July 13th, 1904.

WALTER EDMUNDS,
P. LOCKHART MUMMERY, } *Auditors.*

Meetings of the Society were held during the Session of 1903–1904 *at the undermentioned places.*

1. Friday, October the 16th, 1903.—The Hotel Cecil, Strand. Mr. Clinton Dent in the Chair.
2. Friday, November the 20th, 1903.—No. 11, Chandos Street, Cavendish Square, W. Dr. H. R. Hutton (Manchester) in the Chair.
3. Friday, December the 11th, 1903.—No 11, Chandos Street, Cavendish Square, W. Mr. Sydney Stephenson in the Chair.
4. Friday, January the 15th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Dr. Porter Parkinson in the Chair.
5. Friday, February the 19th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
6. Friday, March the 18th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Dr. Percy Lewis (Folkestone) in the Chair.
7. Friday, April the 15th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Dr. C. O. Hawthorne in the Chair.
8. Friday, May the 6th, 1904 (Special Meeting).—No. 11, Chandos Street, Cavendish Square, W. Mr. Watson Cheyne, C.B., F.R.S., in the Chair.
9. Friday, May the 27th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Mr. Walter Edmunds in the Chair.
10. Saturday, June the 18th, 1904 (Provincial Meeting).—The Medical Library, University College, Bristol. Dr. Theodore Fisher in the Chair.
11. Friday, July the 15th, 1904 (Annual General Meeting).—The Hotel Cecil, Strand. Mr. A. H. Tubby in the Chair. At the conclusion of the meeting the first Wightman Lecture was delivered by Mr. R. Clement Lucas.

LIST OF OFFICE BEARERS, PAST MEMBERS OF COUNCIL, WITH DATES OF SERVICE, AND MEMBERS OF THE SOCIETY FOR THE SESSION 1904—1905.

(*Revised October*, 1904.)

(O.M. = Original Member. T. = Treasurer. S. = Secretary. S with date attached = Past Secretary. P.S. = Provincial Secretary. P.S. with date attached = Past Provincial Secretary. Ed. = Editor of the Reports. C. = Member of the Council. C with date attached = Past Member of Council. * = Life Member. C.C. = Chairman of Council. C.C. with date attached = Past Chairman of Council. W = Wightman Lecturer. W with date attached = Past Wightman Lecturer.

Elected

O.M. Abrahams, Bertram Louis, M.B., Assistant-Physician at Westminster Hospital; 14, Welbeck Street, W.

O.M. Achard, Alexander Louis, M.D., 9, Blandford Street, Manchester Square, W.

1902 Adams, Charles E., 227, Gipsy Road, S.E.

1901 Allingham, Herbert William, Surgeon to H.M. The King; Surgeon at The Great Northern Hospital; Assistant-Surgeon at St. George's Hospital; 25, Grosvenor Street, W.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks.

1903 Anderson, J. Ford, M.D., 41, Belsize Park, N.W. (C.)

1901 Andriezen, William Lloyd, M.D., 7, Apsley Terrace, Acton.

1901 Archdale, Mervyn A., M.B., East Riding Lunatic Asylum, Beverley.

Elected

1902 Armour, Donald John, M.B., Assistant-Surgeon at The West-London Hospital; Senior Assistant-Surgeon at The Belgrave Hospital for Children; 89, Harley Street, W.

O.M. Armstrong, Henry George, Medical-Officer at Wellington College; Wellington College, Berks. (C. 1900—1901.)

1901 Armstrong, John T., Selby House, Beaumont Terrace, West Kensington, W.

O.M. Ashby, Henry, M.D., Physician at The Manchester General Hospital for Children; Lecturer on Diseases of Children at Owens College; 13, St. John Street, Manchester. (C. 1900—1903.) (W.)

O.M. Barnardo, Thomas John, 18, Stepney Causeway, E.

1900 Barr, James, M.D., Physician at The Royal Infirmary, Liverpool; 72, Rodney Street, Liverpool.

1902 Barrett, Alfred Keppel, 2, Peel Place, London, W.

1904 Batchelor, Charles, 1, Fairfield Avenue, Staines, Middlesex.

1901 Battams, J. Scott, 123, Lavender Hill, S.W.

1904 Baumann, E. P., M.D., 38, Harley Street, W.

O.M. Beach, Fletcher, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Winchester House, Kingston Hill, Surrey. (C. 1900—1903.)

1901 Beddard, Arthur Philip, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1904 Bidwell, Leonard, Surgeon at The West London Hospital; 15, Upper Wimpole Street, W.

1900 Blagg, Arthur F., M.D., Physician at The Orthopædic Hospital for Children, Bristol; 28, Caledonia Place, Clifton, Bristol.

1902 Blaikie, James Brunton, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 22, Grosvenor Street, W.

O.M. Bluck, Walter Cardy, M.B., Byford House, York Road, Southend-on-Sea.

O.M. Blumfeld, Joseph, M.D., Assistant-Anæsthetist at St. George's Hospital; 7, Cavendish Place, W.

Elected

1904 Bolton, Charles, M.D., Assistant-Physican at The North-Eastern Hospital for Children, Hackney Road, N.E.; 22, Bentinck Street, W.

1900 Bolus, Harry Boulcott, M.B., "Haddon," Beckenham, Kent.

1903 Braithwaite, John, M.D., Physician at The Devonshire Hospital and Buxton Bath Charity, Hardwick Cliff, Buxton.

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1904 Briscoe, J. Charlton, M.B., Physician to Out-patients at The Evelina Hospital for Sick Children; 18, Harley Street, W.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.B., Hoylake, Cheshire.

1901 Bryant, John Henry, M.D., Assistant-Physician at Guy's Hospital; 8, Mansfield Street, W.

1904 Burgess, Harold, L., North-Eastern Hospital for Children, Hackney Road, N.E.

1904 Burgess, Richard, North-Eastern Hospital for Children.

1901 *Burgess, William Milner, 43, Nicoll Road, Harlesden, N.W. (C.)

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at Paddington-Green Children's Hospital; 86, Harley Street, W.

1904 Burrows, Harold, M.B., Assistant-Surgeon at The Bolingbroke Hospital; 32, Wimpole Street, W.

1901 Burt, Albert Hamilton, 52, Lansdowne Place, Hove, Sussex.

O.M. Burton, Charles George, 77, Church Street, Lower Edmonton, N.

1902 Buzzard, E. Farquhar, M.B., Assistant-Physician at The Belgrave Hospital for Children, Assistant-Physician at The Royal Free Hospital; 33, Harley Street, W.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

1902 Campbell, Henry Johnstone, M.D., Senior Physician at The Bradford Royal Infirmary.

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children; 21, Great Victoria Street, Belfast. (C.)

Elected

1901 Carless, Albert J., Surgeon at King's College Hospital; 10, Welbeck Street, W.

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children; 22, Northumberland Street, Edinburgh. (C. 1900—1902.)

O.M. Carpenter, George, M.D., late Physician at The Evelina Hospital for Sick Children; Assistant-Physician at The North-Eastern Hospital for Children; Editor of 'The British Journal of Children's Diseases'; 12, Welbeck Street, W. (C. 1900—1901.) (S. 1901—1904.) (Ed. 'Reports' 1900—1904.) (Ed.)

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C.)

1900 Cathcart, George C., M.B., Senior Assistant-Surgeon at The London Throat Hospital, 35, Harley Street, W.

O.M. *Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children; Assistant-Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C. 1900—1903.) (S.)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C. 1900—1903.)

1901 Chaldecott, John Henry, Anæsthetist at St. John's Hospital for Diseases of the Skin, at The Metropolitan Throat, Nose, and Ear Hospital, and at The Italian Hospital; 41, Welbeck Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W. (C.)

1901 Cheyne, William Watson, C.B., C.M., F.R.S., Surgeon at King's College Hospital, and at Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901—1902.)

1901 Clapham, E. W., 48, Harley Street, W.

O.M. Clarke, Ernest, M.D., Surgeon at The Central London Ophthalmic Hospital; Ophthalmic-Surgeon at The Miller Hospital, Greenwich; 3, Chandos Street, Cavendish Square, W.

Elected

O.M. Clarke, James Jackson, M.B., Surgeon to Out-patients at The City Orthopædic and at The North-West-London Hospitals; 18, Portland Place, W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collie, Robert John, M.D., Medical-Examiner for The School Board for London; 25, Porchester Terrace, Hyde Park, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 57A, Wimpole Street, W.

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West-London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1901 Cooper, Percy Robert, M.D., "Glenthorne," The Downs, Bowdon, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1901 Cox, Joshua J., M.D., Physician for Diseases of Children to The Clinical Hospital, Manchester; 38, Deansgate, Manchester.

1902 Cross, Ernest, W., "The Limes," Wallwood Park, Leytonstone.

1901 Crossley, Leonard, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 Crowley, Ralph H., M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

1904 Curl, Sydney Walter, M.D., Pathologist at The North-Eastern Hospital for Children; 11, Devonshire Street, Portland Place.

1902 Curtis, Henry J., B.S., late Surgeon at The Royal Hospital for Women and Children; late Surgeon at the North-Eastern Hospital for Children: Surgeon at the Hospital, Buluwayo, S. Africa.

1901 Dawnay, Archibald Hugh Payan, Anæsthetist at The Royal Orthopædic Hospital; 67a, Harley Street, W.

O.M. Deas, Frank, Thorndale, Merton Park, S.W.

Elected

O.M. Dent, Clinton T., M.C., Surgeon at St. George's Hospital and at The Belgrave Hospital for Children; 61, Brook Street, W. (C.)

1901 Dixon, R. Halstead, M.B., 3, Florence Terrace, Ealing Common, W.

O.M. Dockrell, Morgan, M.D., Physician at St. John's Hospital for Diseases of the Skin; 9, Cavendish Square, W.

1901 Dodd, Arthur Herbert, 49, Church Road, Hove, Sussex.

1901 Douglas, Arch. R., Assistant-Medical Officer and Deputy Superintendent at The Royal Albert Asylum, Lancaster.

1901 Drake-Brockman, E. F., Lieutenant-Colonel I.M.S., late Professor of Physiology and Diseases of the Eye at The Medical College, Madras, and late Superintendent at The Eye Infirmary, Madras, India; 14, Welbeck Street, W.

O.M. Drew, Douglas, B.S., Surgeon at The North-Eastern Hospital for Children and at The Hospital for Women, Soho Square; 1, Harley Street, W.

1901 Dun, Robert Craig, M.B., Surgeon at The Liverpool Infirmary for Children; 6, Rodney Street, Liverpool.

1901 Dunlop, James, Aveley, Purfleet, Essex.

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West Derby Road, Liverpool.

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

1904 Edmunds, Arthur, B.S., Assistant-Surgeon at The Paddington Green Children's Hospital; 23, Upper Wimpole Street, W.

O.M. Edmunds, Walter, M.C., Surgeon at The Tottenham Hospital; 2, Devonshire Place, W. (C.1900—1903.)

1901 Ellis, W. McDonogh, M.D., Assistant-Physician at The Hospital for Women, Bath; Cedar Lodge, Bath.

1904 Evans, Arthur, M.S., Assistant-Surgeon at Westminster Hospital, Surgeon to out-patients at The Seamen's Hospital, Royal Victoria and Albert Dock; 53, Queen Anne Street, W.

1901 Evans, J. Howell, M.Ch., 31, New Cavendish Street, Portland Place, W.

Elected

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W. (C.)

O.M. Fenwick, William Soltau, M.D., Senior Physician at The London Temperance Hospital, and Physician at The Evelina Hospital for Sick Children; 29, Harley Street, W.

1901 Fisher, H. Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., Pathologist at The Bristol Royal Infirmary, and Physician to Out-patients at The Bristol Hospital for Sick Children; Harley Lodge, Clifton Down, Bristol. (C. 1900—1901.) (P.S. 1901—1904.) (C.)

1902 Flower, George John William, "Hendford," Yeovil, Somerset.

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

1903 Fortescue-Brickdale, John Matthew, M.D., Registrar and Pathologist at The Royal Hospital for Children and Women, Bristol; 52, Pembroke Road, Clifton, Bristol.

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at Paddington-Green Hospital for Children; 14, Harley Street, W.

1901 Francis, J. E., Charlcote, Northam, North Devon.

1901 Freeland, Ernest Harding, Surgeon at St. George's and St. James's General Dispensary; 145, Harley Street, W.

1901 *Frost, Edmund, M.B., "Chesterfield," Chesterfield Road, Eastbourne.

O.M. Fuller, Andrew, Local Government Board Inspector; "The Bushes," Chase Road, Oatlands Park, Weybridge, Surrey.

1901 George, Alfred Walter, M.D., Surrey House, Shootup Hill, N.W.

1900 Gervis, F. Harcourt, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

Elected

O.M. GIFFARD, HENRY EDWARD, Denham House, Egham.

O.M. GILES, LEONARD THOMASON, 5, St. Nicholas Parade, Scarborough.

1901 GRANT, J. DUNDAS, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. GRAY, ALEXANDER C. E., M.D., 1, Elm Park Gardens, South Kensington, W.

1901 GULLAN, ARCHIBALD GORDON, M.D., 5, Canning Street, Liverpool.

O.M. GUTHRIE, LEONARD GEORGE, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Senior Physician at The Paddington-Green Children's Hospital; 15, Upper Berkeley Street, W. (C. 1900—1903.) (C.)

O.M. GWYNNE, CHARLES NELSON, M.Ch., Surgeon at The Children's Hospital, Sheffield; Highfield House, Sheffield. (C.)

1904 HARKE, S. L., M.B., North-Eastern Hospital for Children.

O.M. HAWKEN, GILES LOCKWOOD LANG, Surgeon at St. John's College, Hurstpierpoint, Sussex.

1901 HAWTHORNE, CHARLES OLIVER, M.D., Physician at The Central London Ophthalmic Hospital; Assistant-Physician at The Royal Hospital for Children and Women; 63, Harley Street, W.

1901 HAYES, THOMAS C., M.D., Professor of Obstetric-Medicine and Diseases of Women at King's College Hospital; 17, Clarges Street, Mayfair, W.

1901 HEYWOOD, CHARLES CHRISTOPHER, M.B., Winter's Buildings, St. Ann Street, Manchester.

1904 HILL, G. WM., M.D., Surgeon for Diseases of the Ear at St. Mary's Hospital; 26, Weymouth Street, Portland Place, W.

1902 HINDS, FRANK, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing. (C.)

1901 HOBHOUSE, EDMUND, M.D., Assistant Physician at The Sussex County Hospital; Physician at The Royal Alexandra Hospital for Children, Brighton; 12, Second Avenue, Hove. (C. 1903—1904.)

Elected

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C. 1901—1904.)

O.M. Hutton, David Mount, M.D., 5, Lulworth Road, Birkdale.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester. (C.)

1901 Huxley, Henry, 39, Leinster Gardens, Hyde Park, W.

O.M. Jaffrey, Francis, Assistant-Surgeon at St. George's Hospital; Surgeon to Out-patients at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C. 1901—1904.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

1904 Jones, A. Ernest, M.D., University College Hospital, Gower Street, W.C.

1901 Jones, E. White, C.M., Surgeon at The Longton Hospital; "Normacot," Longton, Staffordshire.

O.M. Jones, Robert, Surgeon at The Royal Southern Hospital, Liverpool; 11, Nelson Street, Liverpool. (C. 1900—1902.)

1903 Jones, W. Llewellyn, M.D., 58, Thomas Street, Merthyr Tydfil, South Wales.

1901 Kapadia, S. A., M.D., 49, Longridge Road, South Kensington.

1902 Kelson, William Hy., M.D., Assistant-Surgeon at The London Throat Hospital; 15, Old Burlington Street, W.

O.M. Knapton, George, Yew Bank, Bowdon, Cheshire.

Elected

1904 KNIGHT, FREDERICK, M.D., Physician at The Swansea Hospital; 10, Walter Terrace, Swansea.

O.M. LACK, HARRY LAMBERT, M.D., Ear and Throat-Surgeon at The Children's Hospital, Paddington-Green; Surgeon at The Throat Hospital, Golden Square; 48, Harley Street, W.

1901 LACY, ALEXANDER GAIRDNER, "Saumarez," Sunninghill, Ascot, Berks.

1901 LAKE, RICHARD, Surgeon at The Royal Ear Hospital; 19, Harley Street, W.

O.M. LAMBERT, SYDNEY H. A., M.B., Gothic House, Harrow-on-the-Hill.

1901 LANGDON-DOWN, PERCIVAL L., M.B., 47, Welbeck Street, W.

1900 LANGDON-DOWN, REGINALD LANGDON, M.B., 47, Welbeck Street, W.

O.M. LANKESTER, ALFRED OWEN, 5, Upper Wimpole Street, W.

O.M. LAWSON, ARNOLD, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital, and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green 12, Harley Street, W.

O.M. LEE, PHILIP GEORGE, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork.

1901 LESLIE, W. MURRAY, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1904 LETT, HUGH, Ch.B., Surgical Registrar at The London Hospital; 17, Finsbury Circus, E.C.

O.M. LEWIS, PERCY G., M.D., Honorary Medical Officer at The Victoria Hospital, Folkestone, Kent. (C.)

1904 LINGARD, ALFRED, M.B., Imperial Bacteriologist to the Indian Government, Imperial Bacteriological Laboratory, Nainital District, United Provinces, India.

O.M. LISTER, THOMAS DAVID, M.D., Assistant-Physician Royal Hospital for Women and Children, Assistant-Physician Mount Vernon Hospital; 50, Brook Street, W.

Elected

1904 Lister, William Tindall, B.C., Ophthalmic-Surgeon at The Hospital for Children, Great Ormond Street, Assistant-Surgeon at The Central London Ophthalmic Hospital; 30, Queen Anne Street, W.

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

O.M. *Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T.) (W. 1903—1904.)

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 78, Dublin Road, Belfast. (C. 1901—1902.)

1904 McGregor, John, M.D., 15a, Pembridge Crescent, W.

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C. 1902—1903.)

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C. 1901—1904.)

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling. (C.)

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

1901 Makins, George Henry, C.B., Surgeon at St. Thomas's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 47, Charles Street, Berkeley Square, W.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

1904 Marsh, J. J., 104, Sloane Street, S.W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C. 1900—1903.)

Elected

1900 May, W. Page, M.D., 9, Manchester Square, W.

1902 Maynard, John Seymour, M.B., 14, Charles Street, St. James's.

1904 Midelton, William John, 72, Charminster Road, Bournemouth.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull.

1902 Miller, Charles Hewitt, Oughtrington House, Lymm, Cheshire.

1902 Milligan, Wyndham Anstruther, C.M., Anæsthetist at The Great Northern Central Hospital; 104, Bethune Road, N.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Surgeon at The Northern Hospital, Liverpool; 11, Rodney Street, Liverpool. (C.)

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester, Assistant-Surgeon at The Manchester Royal Infirmary; 15, St. John Street, Manchester.

1901 Morgan, Cecil Augustus, M.B., 6, Somerset Street, Portman Square, W.

1902 Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.

1901 Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W. (C.)

1901 Morton, Samuel Ernest, 72, Burngreave Road, Sheffield.

O.M. Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford.

1902 Mummery, John Percy Lockhart, B.C., 10, Cavendish Place, W., Assistant-Surgeon at The North-Eastern Hospital for Children. (C.)

1901 Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.

Elected

1904 Murphy, T. Keogh, B.C., Surgeon to Out-patients at The Paddington Green Children's Hospital; 16, St. Petersburgh Place, W.

1901 Murtz, Anton Peter, Clareville, 48, Queen's Road, Finsbury Park, N.

1901 Nabarro, David N., M.D., Assistant-Professor of Pathology and Lecturer on Bacteriology, University College; 6, Crown Office Row, Inner Temple, E.C.

1904 Nash, Elwin H. T., Oulton House, Burnley Road, Accrington, Lancs.

1901 Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.

1902 Newbolt, George P., Surgeon at The Royal Southern Hospital, Liverpool; 42, Catherine Street, Liverpool.

1901 Nisbet, J. Tawse, M.D., 7, Croxteth Road, Liverpool.

1901 Niven, Robert, Bethnal House Asylum, Cambridge Road, N.E.

1902 Northcote, Percy, M.B., 101, Jermyn Street, S.W.

1901 Oldmeadow, Lloyd J. H., M.D., Kineton, Warwickshire.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1904 Page, H. M., 14, Greville Place, S.W.

1901 Palmer, Frederick S., M.D., 7, Gordon Place, Gordon Square, W.C.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital and Physician at The North-Eastern Hospital for Children; 57, Wimpole Street, W. (C.)

1902 Parkinson, Thomas W., M.D., 77, Sloane Street, S.W.

1903 Parry, Robert H., Surgeon at The Victoria Infirmary and at The Royal Hospital for Sick Children, Glasgow; 3, Royal Crescent, Queen's Park, Glasgow. (C.) (1903—1904.)

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1903 Pearson, S. Vere, M.B., Assistant-Physician at The East London Hospital for Children, Shadwell; 8, Serjeant's Inn, E.C.

Elected

1901 Pedley, R. Denison, Dental-Surgeon at The Evelina Hospital for Sick Children, S.E.; 17, Railway Approach, London Bridge, S.E.

1901 Pendlebury, Herbert S., B.C., Surgeon at The Royal Hospital for Women and Children, Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. *Penrose, Frank, M.D., Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; 84, Wimpole Street, W.

1901 Pernet, George, Assistant at the Skin Department, University College Hospital; 152, Harley Street, W. (C.)

1901 Perry, Herbert, M.D., Assistant-Physician at The General Hospital, Birmingham, Physician to Outpatients at The Children's Hospital, Birmingham; 54, Newhall Street, Birmingham.

O.M. Peskett, A. W. Chalmers, M.B., 4, Clermont Road, Preston Park, Brighton.

1902 Phillips, Hugh, M.B., 28, Palace Court, W.

1901 Poland, John, Senior Surgeon at The Miller Hospital, Greenwich; Surgeon at The City Orthopædic Hospital; 2, Mansfield Street, W.

1902 Potter, Bernard Elwell, M.B., Resident Medical Officer at St. George's Hanover Square Dispensary; 16, Little Grosvenor Street, W.

1901 Potts, William A., M.D., 160, Hagley Road, Birmingham.

O.M. *Prangley, Henry J., Tudor House, Anerley, S.E.

1901 Pratt, Eldon, M.D., Henfield, Sussex.

1901 Pritchard, George Eric Campbell, M.D., 14, Cromwell Place, South Kensington, S.W.

1901 Quennell, Arthur, Brentwood, Essex.

1901 Raw, Nathan, M.D., Mill Road, Liverpool.

1901 Ray, John Howson, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 22, St. John Street, Manchester.

1901 Reeves, Henry A., Surgeon at The Hospital for Women and at The Royal Orthopædic Hospital; 70A, Grosvenor Street, W.

Elected

1901 Rendel, Arthur Bowes, M.B., 43, Albion Street, Hyde Park, W.

1901 Rhodes, John Milson, M.D., Didsbury.

1903 Ridley, J. Brooke, M.D., 32, Sydenham Road, Croydon.

O.M. Roberts, David Lloyd, M.D., Consulting-Obstetric-Physician at The Manchester Royal Infirmary; 11, St. John Street, Manchester.

1901 Roberts, George A., Oaklands, Langton, Tunbridge Wells.

O.M. Robinson, Henry Betham, M.S., Assistant-Surgeon at St. Thomas's Hospital and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C.)

O.M. Rogers, Bertram, M.H., M.D., Physician at The Hospital for Sick Children and Women, Bristol; 1, Victoria Square, Clifton, Bristol.

1901 Rose, Samuel Frank, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The North-Eastern Hospital for Children; 59, Queen Anne Street, W.

O.M. Roth, Bernard, Orthopædic-Surgeon at The Royal Alexandra Hospital for Children, Brighton; 38, Harley Street, W.

1901 Rowland, Penry W., M.B., Selwood, Crescent Road, Crouch End, N.

1902 Rowlands, Robert Pugh, B.S., The College, Guy's Hospital, Assistant Surgeon at The East London Hospital for Children.

1902 *Roxburgh, Alex. Bruce, B.Ch., Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square, W.

O.M. Rugg, George L., 106, Knight's Hill Road, West Norwood, S.E.

1901 Rumboll, Sydney, Cavendish House, Leeds.

1904 Sale-Barker, Horace John Maurice Drummond, Medical Officer in Charge X-ray and Electrical Departments at The North-Eastern Hospital for Children, also at Westminster Hospital; 49, Weymouth Street, Portland Place, W.

Elected

O.M. *Sansom, A. Ernest, M.D., Consulting-Physician at The London Hospital, and Consulting-Physician at The North-Eastern Hospital for Children; 84, Harley Street, W. (C.C. 1900—1902.) (C. 1902—1903.)

O.M. Sansom, Henry Arthur, M.D., 127, West End Lane, N.W.

1904 Sawyer, James E., M.D., 93, Cornwall Street, Birmingham.

1901 Schilling, George, 41, Tollington Park, N.

1904 Scott, W. J., M.B., Clifton House, Sheet Street, Windsor.

O.M. Sequeira, James H., M.D., Physician in Charge of the Skin Department at The London Hospital; 63, Harley Street, W.

1901 Shackleton, William Webb, M.B., Grove House, Bushey, Herts.

1901 Shenton, Edw. W. H., 20A, Hanover Street, Hanover Square, W.

O.M. Shuttleworth, George E., M.D., late Medical Superintendent Royal Asylum for Imbeciles, Lancaster; Medical Examiner defective children, Willesden School Board; Medical Expert (M.A.B.) Rochester House Inst., Ealing; Ancaster House, Richmond Hill, Surrey. (C. 1900—1903.)

1901 Simson, Henry J. Forbes, 80, Brook Street, W.

1901 Skelding, Henry, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford. (C.)

1901 *Smith, Frederick John, M.D., Physician at The London Hospital; 138, Harley Street, W.

1901 Smith, Guy Bellingham, B.S., Surgeon at The Surrey Dispensary; 21, St. Thomas's Street, S.E.

1901 *Smith, Travers, R.M., M.B., 13, Kilburn Park Road, N.W.

1901 Smyth, Sidney B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast. (C.)

O.M. Snape, Ernest, M.D., 41, Welbeck Street, W.

O.M. Spokes, P. Sidney, Dental-Surgeon at University College Hospital; 4, Portland Place, W.

Elected

1903 Spriggs, Edmund Ivens, M.D., Physician to Out-patients at The Victoria Hospital for Children and at The City of London Chest Hospital; 24, St. Thomas's Street, S.E.

1904 Spurway, John, M.B., 29, Brondesbury Road, Kilburn, N.W.

O.M. Staunton, Michael Cornelius, M.D., Visiting-Surgeon at The Children's Hospital, Dublin; 22, North Frederick Street, Dublin.

1901 Staveley, William H. C., 24, Sloane Gardens, S.W.

O.M. Stephenson, Sydney, C.M., Ophthalmic-Surgeon at The Evelina and The North-Eastern Hospitals for Children; Editor of *The Ophthalmoscope*; 33, Welbeck Street, W. (S. 1900—1903.) (C. C. 1903—1904.) (C.C.)

1901 Steward, Francis James, M.S., Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; Assistant-Surgeon and Surgeon in charge of the Throat Department at Guy's Hospital; 133, Harley Street, W.

O.M. Stewart, Hugh M., M.D., Walton House, Lordship Lane, S.E.

O.M. Stiles, Harold J., C.M., Surgeon at The Royal Edinburgh Hospital for Sick Children; 8, St. Colme Street, Edinburgh. (C. 1900—1901.) (C. 1902—1904.)

1900 Street, Alfred Francis, M.D., "Burghfield," St. Mildred's Road, Westgate-on-Sea.

O.M. Summerhill, Thomas Henry, Surgeon at The Alexandra Children's Hospital, Rhyl; "Bodfor," Rhyl, Flintshire.

O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; Obstetric-Physician at the French Hospital; 11, Cavendish Place, W.

O.M. Sutherland, George A., M.D., Physician at The Paddington-Green Children's Hospital; Assistant-Physician at The North-West-London Hospital; 73, Wimpole Street, W. (C. 1900—1903.) (S.)

1904 Swainson, James Midleton Graham, North-Eastern Hospital for Children.

1901 Swan, Russell H. J., M.B., 8, St. Thomas's Street London Bridge, S.E.

Elected

1901 Swanton, James Hutchinson, M.D., 40, Harley Street, W.

1901 *Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 19, Upper Wimpole Street, W.

1901 *Taylor, Frederick, M.D., Senior Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901—1902.) (C. 1903—1904.)

O.M. Taylor, James, M.D., Physician at The National Hospital for Paralysed and Epileptic, Queen Square, and Physician at The North-Eastern Hospital for Children; 49, Welbeck Street, W. (C. 1901—1904.)

1902 Thorne, Atwood, M.B., Assistant-Surgeon at The London Throat Hospital; 148, Harley Street, W.

1901 Thorne, W. Bezly, M.D., 53, Upper Brook Street, W.

1902 Tod, Hunter, B.C., 101, Harley Street, W., Aural-Surgeon at The London Hospital.

1901 Trout, J. H. Ensor, 412, Monument Road, Birmingham.

O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 25, Weymouth Street, W. (C. 1900—1902.) (C.C. 1902—1903.) (C.)

1903 Tullis, George Augustus, M.D., Holmbush, Grove Road, Southsea.

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Square, W.

1901 Turner, H. Stanley, "Balgownie," 211, Bridge Road, Battersea, S.W.

1904 Turner, Philip, M.S., Assistant-Surgeon at The Belgrave Hospital for Children; 8, St. Thomas's Street, S.W.

1904 Turton, Edward, M.D., Physician at the Leeds General Infirmary; Clifton House, Goole, Yorks.

1902 Turton, James, "Hatherley," Preston Park, Brighton.

O.M. Vacher, Francis, Consulting-Surgeon at The Wirrell Hospital for Children; "Glenmore," Shrewsbury Road, Birkenhead.

O.M. Vickery, William H., Surgeon at The Children's Hospital, Newcastle-on-Tyne; 52, Elswick Road, Newcastle-on-Tyne. (C. 1900—1901.)

Elected

1902 Walker, J. W. Thomson, C.M., Assistant-Surgeon at The North-West London Hospital, Assistant-Surgeon at St. Peter's Hospital for Stone; 8, Cavendish Place, W. (C.)

1901 Wallace, Joseph B., M.B., 39, Battersea Rise, S.W.

1901 Walsh, David, M.D., Physician at The Western Skin Hospital; Editor of 'The Medical Press and Circular'; 70A, Grosvenor Street, W.

1901 Waring, John Alfred, M.B., 21, Derby Road, Nottingham.

1901 *Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W.

1902 Westmacott, Frederick H., Aural-Surgeon at the Manchester Children's Hospital, Pendlebury; 8, St. John Street, Manchester.

1901 Whipham, Thomas Rowland C., M.B., Assistant-Curator of the Museum at St. George's Hospital, Physician to Out-patients at the Evelina Hospital for Children; 37, Park Street, W.

1901 Whitelocke, Richard H. A., C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C. 1901—1904.) (P. S.)

O.M. Wilks, Joseph Henry, B.C., Surgeon at The Children's Hospital, Sheffield; "Fairfield," Sharrow, Sheffield.

O.M. *Williams, Dawson, M.D., Physician at The East-London Hospital for Children, Editor of 'The British Medical Journal'; 2, Agar Street, Strand, W.C. (C. 1900—1902.)

O.M. Williams, Edward Cecil, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 9, Whatly Road, Clifton, Bristol.

O.M. Willey, Charles H., C.M., Surgeon at The Sheffield Children's Hospital; Wostenholm Road, Sharrow, Sheffield. (P.S. 1900—1901.) (C. 1901—1902.)

1904 Willis, Charles Faircourt, Lieut.-Colonel, I.M.S., M.D., "Felixstowe," The Avenue, Upper Norwood, S.E.

1901 Wills, William Alfred, M.D., Assistant-Physician at Westminster Hospital; Senior Physician at The North-Eastern Hospital for Children; 29, Lower Seymour Street, W. (C.)

Elected

1904 Wilson, Geoffrey R., Ongar, Essex.

O.M. Wilson, Kemlo R. M., M.D., Hanwell, W.

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*** *It is requested that any inaccuracy in the foregoing list may be notified to the Honorary Editor of the Reports and to either of the London Honorary Secretaries.*

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REPORTS

OF

THE SOCIETY

FOR THE

STUDY OF DISEASE IN CHILDREN.

SESSION of 1904–5.

1. STRANGULATED INGUINAL HERNIA IN AN INFANT TWENTY-EIGHT DAYS OLD; OPERATION TWO DAYS LATER; RECOVERY.

By R. CLEMENT LUCAS, B.S.

The following report was made by Mr. H. H. Mayle, the Ward Clerk.

J. E—, who was born on June the 27th, 1903, was admitted into Guy's Hospital on July the 27th, 1903, suffering from a strangulated right inguinal hernia.

He was the first child of healthy parents and had been breast fed. He grew fat and looked well, but the mother stated that he frequently seemed in pain, often crying when passing urine or fæces, and sometimes vomiting part of the milk taken.

On the evening of Saturday, July the 25th, when being washed, the child's scrotum was noticed to be swollen.

The same night he was seized with a violent fit of screaming and vomited several times. A doctor was called in the next morning, but vomiting continued all day and the following night, and on Monday, July the 27th, the medical man advised the parents to bring the child to the hospital. His bowels had not acted for two days, and he had vomited everything he had taken since Saturday. The mother declared he had not passed water since the symptoms commenced.

When admitted into the hospital he appeared to be a plump, healthy child, suffering from pain and sickness, with a distended abdomen.

On examining the scrotal region three distinct swellings were observed. The scrotum itself was dark blue in colour, firm, and distended. Above this, in the right inguinal region, another tense swelling was found. There was a distinct interval between the two, in which the cord could be felt. The left half of the scrotum was empty, but the left testis could be felt just outside the external abdominal ring forming the third swelling.

Mr. Clement Lucas, on examining the case, determined to explore the upper swelling on the right side at once, thinking this might be a strangulated funicular hernia. The swelling in the scrotum, which was tense and dark, he attributed to serous effusion and blood, partly as the result of pressure above and partly due to taxis. On the left side the testis had not descended into the scrotum, and was probably associated with an unclosed vaginal process of peritoneum into which some fluid from the abdomen, which quickly follows obstruction in infants, had probably made its way.

The operation was performed about 3.30 p.m. on July the 27th. A.C.E. mixture, and subsequently chloroform, were administered, and Mr. Lucas cut down upon the right inguinal swelling, making an oblique incision in the direction of the inguinal canal. When the skin and superficial fascia had been divided, the swelling exposed was dark blue in appearance. To explore its relation to the testis

the right testis was pushed up into the wound and the tunica vaginalis showed blood extravasation, as if the result of taxis. The two swellings were not continuous except so far as the cord connected them. Picking up the sac it was divided with scissors, when some blood-stained serum escaped, and at the bottom a small knuckle of intestine was exposed. This was about the size of the end of one's forefinger, and dark purple in colour, but it had not lost its lustre. A blunt dissector was passed up by the side of the bowel, and by drawing the instrument forwards the neck of the sac was stretched, after which the bowel was, by gentle pressure, slipped back into the abdomen. Some drachms of clear serous fluid escaped from the abdomen after the bowel had been replaced. The sac was tied and cut away, and two buried silk sutures were put across the canal and ring to complete a radical cure. A continuous horse-hair suture was used for the skin. A sealed dressing of gauze, collodion, and iodoform was applied to protect the wound, and cyanide dressings over.

The patient's temperature rose to 103·6° F. the same night, but fell to normal on the following day, and never again rose higher than 99° F. whilst in the hospital. His bowels acted freely during the night and vomiting ceased after midnight.

July the 30th.—The patient was dressed for the first time, and the scrotum showed signs of irritation, due probably to the antiseptic dressings. The temperature varied during the day from 98° F. to 99° F. Boracic fomentations were applied.

August the 7th.—The stitches were removed, as they were causing slight superficial suppuration. Temperature had continued normal.

August the 9th.—The boracic fomentations were discontinued and dry dressings applied.

August the 11th.—A sealed collodion and gauze dressing was applied.

August the 12th.—The wound being soundly healed and the child in excellent health, he was to-day discharged.

Remarks.—Strangulated hernia in infants is of special interest not only from its comparative rarity, but for the reason that it is very liable to be overlooked. Vomiting and colic from injudicious feeding are so common that the graver condition of hernial strangulation may be easily confounded therewith. The relative proportion of strangulation in children compared with adults is given by C. Stern* as 1 to 108, which is easily accounted for by the greater elasticity of the tendinous and fibrous structures in young children. It will be noticed that the form of hernia present in the case related was that known as "funicular," the upper part of the vaginal process of peritoneum into which the hernia had descended being shut off from the tunica vaginalis of the testis. Howard Marsh† relates a similar case in the 'St. Bartholomew's Hospital Reports' for 1874, and gives a table of forty-seven cases of strangulated hernia occurring in children under the age of fourteen. Of these all except four were herniæ into congenital sacs. Two of the exceptions were cases of the "Infantile hernia of Hey," and two others did not communicate with the tunica vaginalis, and were therefore probably funicular. Of twenty-five in which the side was mentioned nineteen were on the right and only six on the left.

The first series of cases of strangulated hernia in children appears to have been collected by Rovoth‡ in 1858, who put on record 30 cases. Sariel,§ in 1894, collected 128 cases. Knoblach and Stern‖ collected 153 cases, of which 138 were inguinal, 14 umbilical, and 1 femoral. Moynihan,¶ who published a case in 1897, concluded from the foregoing researches that strangulation was more common in the earlier months of childhood than

* 'Centralblatt für Chirurgie,' 1894.

† 'St. Bartholomew's Hospital Reports,' 1874.

‡ 'Die herniatomie bei kindern. Deutsche klinik,' No. 29, 1858.

§ 'Revue mensuelle des maladies de l'enfance,' May, 1894.

‖ 'Centralblatt für Chirurgie,' 1894.

¶ 'Lancet,' September 25th, 1897, vol. ii, p. 789.

in the later, as shown by the following table of cases occurring during the first twelve months:

During the	1st	month	16	cases	occurred.
„	2nd	„	15	„	„
„	3rd	„	14	„	„
„	4th	„	9	„	„
„	5th	„	4	„	„
„	6th	„	7	„	„
„	7th	„	3	„	„
„	8th	„	6	„	„
„	9th	„	9	„	„
„	10th	„	3	„	„
„	11th	„	3	„	„
„	12th	„	4	„	„

He also drew attention to the fact that retention of urine is frequently mentioned as present.

The seat of strangulation is in a majority of cases at the neck of the sac; thus Sariel found it in this situation in 58 out of 81 cases examined. The next point of frequency for the constriction is at the external abdominal ring. Marsh found it there in 7 out of 32 cases in which the site was mentioned.

Cases of strangulated hernia in infants under one year of age have been recorded in this country by Macnamara,* Beck,† Dobbin,‡ Haslewood,§ Steele,‖ D'Arcy Power,¶ Kidd,** Maclaren,†† and MacLaurin.‡‡

The symptoms almost invariably commence in infants with a violent fit of screaming which cannot be pacified, indicating that the child is suffering agony. This form of commencement is usually present in other forms of

* 'Lancet,' vol. ii, 1879, p. 505.
† 'Brit. Med. Journ.,' vol. ii, 1882, p. 1151.
‡ 'Lancet,' vol. i, 1884, p. 979.
§ 'Lancet,' vol. ii, 1884, p. 1171.
‖ 'Lancet,' vol. ii, 1899, p. 279.
¶ D'Arcy Power, 'Lancet,' vol. ii, 1899, p. 889; and vol. i, 1901, p. 1536.
** 'Lancet,' vol. ii, 1899, p. 1016.
†† 'Lancet,' vol. ii, 1899, p. 1094.
‡‡ 'Lancet,' vol. 1, 1900, p. 1281.

obstruction, such as intussusception, from which the presence of an irreducible hernial tumour devoid of impulse sufficiently distinguishes it. Vomiting rapidly follows, and persists or recurs whenever the infant is put to the breast or fed. Constipation is usually not noticed till some hours later, and then the mother frequently notices another change—that the diapers are seldom, if at all, moistened by urine. Suppression or great diminution in the amount of urine passed, when occurring in adults during obstruction, is generally accepted as indicating that the constriction is high up in the small intestine. This does not appear to be a sufficient explanation of the cases under discussion. It is possible that spasmodic retention may account for a limited number in whom reduction is early effected; but the general depression of circulation and tendency to collapse, which rapidly supervenes upon strangulation in infants, probably accounts better for what is a suppression of urine rather than a retention.

Another point to which I wish particularly to draw attention is the rapid effusion of a large quantity of serous fluid into the sac, so as to render it for the most part transparent. This has more than once led to an error in diagnosis, and the sacs have been tapped as hydroceles. This serous effusion is not confined to the sac of the hernia, the general peritoneal cavity sympathises and pours out fluid as the case goes on. It will be noticed in the case related, that in addition to the serous effusion into the funicular sac which contained the bowel, there was a large collection in the tunica vaginalis below forming a tumour of considerable size quite separate from the sac, and further, that the fluid escaping from the abdominal cavity into a congenital sac associated with an undescended testis on the left side, formed a third tumour in the left inguinal region.

Another symptom much more marked in infancy than in adults is the early tendency to collapse. This, again, is easily accounted for by the necessity in early infancy of

frequent feeding and the rapid loss of nutrition dependent on the persistent vomiting. Pain, too, which is ill-borne by infants, doubtless contributes to the collapse. When, however, relief has been afforded, the natural recuperative force of childhood will in the majority of cases, where asepsis can be maintained, bring about a rapid recovery.

To sum up the symptoms peculiar to infants as compared with adults: firstly, there is the violent screaming fit impossible to pacify; secondly, the tendency to suppression of urine as well as constipation; thirdly, the rapid effusion of serous fluid into the sac, rendering it translucent, and effusion into the general peritoneal cavity; and fourthly, the greater tendency to rapid collapse.

(*Read October the* 16*th*, 1903.)

Discussion.

Mr. Henry Skelding (Bedford) said since these cases are somewhat infrequent he would like to record a case on which he had operated successfully a few weeks before. The symptoms and history were very similar to Mr. Lucas's case. The child was 7 weeks old, but not too well nourished. There was a considerable quantity of clear serous fluid in the sac, as noted by Mr. Lucas; a portion of the large intestine was constricted by the neck of the sac. He would like to ask Mr. Lucas what portion of the bowel was strangulated in his case; for when operating for radical cure in an infant some weeks ago, where the mother assured him that although the truss always seemed to fit, and no portion of the bowel came down, the child had frequent fits of screaming which did not cease until the truss was removed, he found the sac contained a long thin appendix $4\frac{1}{2}$ inches in length. After removal of the appendix the child made a complete recovery. Regarding the incision he would be glad to know whether Mr. Lucas considered the usual incision satisfactory, or whether he had any difficulty with the testis being drawn up into the wound and getting in the way. The method he, Mr Skelding, had lately adopted was, starting from over the external ring, to carry the incision outwards and upwards towards the crest of the ilium, so as to approach the sac as it emerged from the ring. The advantages claimed were: (1) easier manipulation without drawing up the testis; (2) less friction on the wound and the truss; (3) dressings less liable to soiling. Certainly his late results justified a continuance of these methods.

Mr. J. W. Thomson Walker said Mr. Lucas had mentioned that from the large amount of effusion of serum into the sac a diagnosis of hydrocele had frequently been made and the swelling tapped. He had seen a case in which the opposite mistake was made; a large scrotal swelling in a child was accompanied by vomiting, pain, and constipation, and a diagnosis of strangulated hernia was made. The case, however, turned out to be one of acute hydrocele of the tunica vaginalis. He should be glad to hear what Mr. Lucas' experience had been in regard to the frequency of such cases.

Mr. Jackson Clarke mentioned the case of a new-born baby that was deeply jaundiced, vomited frequently, and presented two large scrotal herniæ. One of the latter was readily reduced, the other could not be reduced, and there being no abnormality about the anus, Mr. Clarke did herniotomy and returned the bowel to the abdomen. The vomiting continued and the child died, and on examination the condition was found to be an interruption in the small intestine at the site of Meckel's diverticulum.

Mr. Clement Lucas, in reply to Mr. Skelding, said that it was a portion of small intestine that was found in the sac. He had not found any inconvenience from an oblique incision over the canal and external abdominal ring, but thought it unwise to extend it into the scrotum. In answer to Mr. Thomson Walker, he said that acute hydrocele might sometimes be accompanied by symptoms suggestive of strangulated bowel, especially if there were peritonitis with an open vaginal process of peritoneum, but in any such case opening of the vaginal process would be for the benefit of the patient.

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2. REMARKS UPON "ETHYL CHLORIDE" AS A GENERAL ANÆSTHETIC."

By J. HENRY CHALDECOTT.

It is comparatively seldom that innovations of any importance occur in the province of anæsthetics, at any rate so far as the introduction of new anæsthetic agents is concerned. Occasionally a new mixture or substance makes its appearance, but after a short trial is discarded, having failed to hold its own with its older rivals, nitrous oxide, ether, and chloroform.

It seemed at one time as if ethyl chloride would, in this country at any rate, have met with a similar fate, but chiefly owing to the perseverance of Dr. MacCardie, of Birmingham, its claims began to be recognised, and I venture to think that before very long it will be generally used for operations of a certain kind.

My own experience of its use dates from about two years ago, when I gave it an extensive trial for operations upon the throat and nose. I was at that time administering it from a Brener's inhaler, but I found it so unreliable, and the induction of anæsthesia in many cases so tedious, that for a time I discarded the drug entirely. I subsequently, however, ascertained that Dr. MacCardie, who had just published a long list of successful cases, administered it from an Ormsby's ether inhaler, and since adopting this method, I have had nothing but unqualified success with it, and have come to regard it as an almost ideal anæsthetic for suitable cases.

There are three methods of using ethyl chloride to which I should like to call attention.

No. 1 consists of cases in which a single dose only is given, and anæsthesia having been induced, the mask is removed, and the operation performed. This class of case includes all short operations upon the nose and mouth, such as tonsillotomy and the removal of adenoids, and as a very large percentage of these cases are children, this branch of the subject is of special interest to us as members of this Society. As you are probably aware, there has for some years been a wide difference of opinion as to the proper anæsthetic to employ for this operation, one school maintaining that chloroform is the only suitable agent, and refusing to be reconciled to the use of nitrous oxide, or to the mixture of nitrous oxide and ether, on the ground that they do not give a satisfactory type of anæsthesia, and that by exciting cyanosis and secretion of mucus they embarrass the operator. The "gas and ether" school, on their side, draw attention to the enormous mortality which seems to be inseparable from the use of chloroform

in these cases, and contend that it is unjustifiable to subject patients to such a risk for an operation which in itself entails no danger to life.

Now, although I belong to the latter party, I am quite prepared to admit that, especially in the case of children, nitrous oxide or nitrous oxide and ether anæsthesia is by no means always ideal, and I am in consequence doubly glad to welcome the substitution of ethyl chloride on the ground that it strikes the happy medium by combining the respective good points of chloroform, nitrous oxide, and ether, without any of their attendant disadvantages; it produces, in fact, a type of anæsthesia as quiet and as free from asphyxial symptoms as the best form of chloroform narcosis, and at the same time is as rapid as nitrous oxide and as stimulating as ether.

Its points of superiority over chloroform in these cases are:—(1) It can be given with the patient in any position; (2) anæsthesia can be induced very much more rapidly than would be possible, or at any rate safe, with chloroform; (3) there is no struggling; (4) a measured dose can be given; (5) it is probably much safer; (6) the after-effects are quite trifling, or absent altogether.

Its advantages over nitrous oxide are:—(1) The anæsthesia is of a better type in that it is quieter, there being a complete absence of suffocative symptoms, such as opisthotonos and jactitation; (2) no cumbrous apparatus is necessary; (3) the available anæsthesia is about twice as long.

Its advantages over ether are:—(1) It is much pleasanter to take; (2) induction of complete anæsthesia is much quicker, and is unattended by struggling; (3) there is no cyanosis or secretion of mucus; (4) it does not leave an unpleasant taste in the mouth or smell in the room; (5) the after-effects are generally much less.

In administering ethyl chloride for these or for other short operations upon the mouth, such as dental extractions, the length of available anæsthesia can be estimated with tolerable accuracy by the dose given, and one can

consequently arrange matters according to the pace which may be expected of the particular operator—but in this connection my experience leads me to utter a word of warning, viz. that if for the removal of tonsils and adenoids it is thought necessary to push the drug so as to obtain an anæsthesia of more than forty seconds, it is safer to have the child lying down, and the fauces well sponged out as the bleeding occurs. When nitrous oxide, either by itself or combined with ether is used, this is rarely necessary, as the swallowing reflex remains fairly active, but with ethyl chloride it disappears more suddenly and returns much more slowly, and unless care is taken blood may find its way into the air passage.

2. The second method of administration is for short cases, which, from the position of the part operated upon, do not necessitate the removal of the mask, and this class includes practically all short minor operations.

It is difficult just yet to speak with absolute certainty as to how ethyl chloride compares in point of safety with the other anæsthetics, but it certainly appears to be safer than chloroform; and if further experience does not disillusionise us upon this point, it might well supersede chloroform in all minor operations. It is notorious how many of the chloroform fatalities occur during minor operations, and one can imagine nothing more distressing than for a child to lose its life for the sake of undergoing some slight surgical procedure, which *per se* carries no risk.

I have administered ethyl chloride for opening abscesses, removing foreign bodies from noses and ears, tenotomy, circumcision (one case was a child of fourteen days old), rectal and abdominal examinations, amputation of finger, and osteotomy (tibia), and have never yet met with any alarming symptoms. Used in this way for short surgical emergencies, ethyl chloride should prove a great boon to the general practitioner, who has perhaps on the spur of the moment to deal singlehanded with a fractious and struggling child. Two cubic centimetres sprayed on to the sponge, and the mask held tightly on the face, will in

a few seconds produce a sufficient anæsthesia for the opening of an abscess, or for an examination of an injured joint or limb. In the necessarily hurried work of the casualty departments of hospitals, ethyl chloride is immensely useful.

The after-effects in these cases are of course more marked than in "single dose" cases, but unless the patient's stomach is full of food, there is rarely severe vomiting.

3. The third method of administration consists of inducing anæsthesia by means of ethyl chloride, and continuing it with ether or chloroform according to the requirements of the case, and for this purpose the drug is very useful on account of the rapidity of induction and the absence of early cyanosis and struggling.

A mixture known as "Somnoform," of which ethyl chloride is the principal ingredient, has lately been extensively used. It has not, however, any single advantage over the simple drug, and compares unfavourably with it in smelling nasty and leaving an unpleasant garlicky taste in the mouth; nor is there, so far as I can see, any advantage in using a mixture of drugs when a single one answers the purpose equally well, if not better.

The actual administration of ethyl chloride is very easy when an Ormsby's inhaler is used, and I believe this is the best and simplest form of apparatus. The only slight modification which I have had added to the ordinary "Ormsby" is to have the mask rather deeper than usual, so that the sponge may not be too near to the patient's nose. With this mask I never meet with any coughing or struggling, and most of my patients tell me afterwards that the inhalation was by no means unpleasant.

In the case of a child two to three c.c. of the drug are sprayed on to the sponge, the mask is applied tightly to the face, and one breath allowed with the air-slot open, the slot is then closed, and no further air admitted until anæsthesia is complete; snoring usually ensues within twenty seconds, and a few seconds later the patient will be perfectly anæsthetic, with flaccid limbs and insensitive

cornea. The mask can now be removed and the operation performed.

If the operation is one which does not necessitate the removal of the mask, it can be commenced after two or three snoring respirations, and if it is wished to continue the anæsthesia with ethyl chloride, more of the drug must be sprayed on the sponge from time to time as required. In these cases it is as well to have a second sponge at hand, to replace the first when it gets frozen. I always use Duncan and Flockhart's pure ethyl chloride.

I have now administered ethyl chloride over five hundred times, and the conclusions I have arrived at are:—(1) It is the best anæsthetic known for what may be called "single dose" cases, when the patient is anæsthetised, the mask removed, and the operation performed without any further administration; (2) it is certainly worthy of an extensive trial in all minor operations; (3) it is a safe and rapid means of inducing anæsthesia as a preliminary to chloroform or ether narcosis; (4) although its effects are transient it is doubtless a powerful anæsthetic, and should be carefully administered, and the patients properly prepared. (*Read October the 16th*, 1903.)

Discussion.

Mr. Atwood Thorne congratulated Mr. Chaldecott on his success. Mr. Thorne had only had the anæsthetic given for him some twenty or thirty times, for adenoids or similar operations. Of those cases, two had been insufficiently anæsthetised and one (who had perhaps swallowed some blood) suffered from severe vomiting and had to be taken into the hospital. Mr. Thorne had heard that there was considerable danger in the drug and wished to know more about this. He certainly had found the anæsthetic very convenient.

Mr. Sydney Stephenson had had considerable experience in ophthalmic out-patient work of ethyl chloride anæsthesia and was more than satisfied with it. The anæsthesia was quickly induced and passed off quickly, and he had never witnessed any untoward symptom. Vomiting, he thought, was generally due to an overdose of ethyl chloride. It had, he considered, great advantages in eye work.

3. A NOTE ON PARALYTIC CHOREA.

By J. PORTER PARKINSON, M.D.

At a meeting of this Society, held earlier in the year, a patient was shown by Dr. Frederick Taylor, described as suffering from "heart disease and athetosis." The involuntary movements were confined to one side of the body, and were accompanied by considerable weakness. A most interesting discussion followed between Dr. Taylor and another member, who suggested that the case was one of hemichorea, and who brought forward many arguments in favour of this diagnosis. It may be then of interest if I relate a case which was under my care in the wards of the Temperance Hospital.

The patient, a boy aged 10 years, was admitted with great paresis, almost complete paralysis of the right side of the body, so that he was unable to walk, dress, or feed himself.

The history was indefinite, but he had apparently been suffering for two weeks from this complaint.

On admission he was a rather thin but fairly well grown boy with average intelligence, but somewhat emotional. There was marked weakness of the right arm and leg, and to a lesser extent of the right side of the face, accompanied by moderately rapid involuntary movements of a purposeless character in the limbs, especially the arms and hands of the paretic side. The heart was normal in size, the apex beat being in the nipple-line; but there was a systolic murmur to be heard at this spot and it was audible outwards for a couple of inches. There was nothing else abnormal about the child, whose reflexes were normal and equal on the two sides.

There were two different opinions possible with regard to this case—Was it one of hemiplegia with athetosis, or was it a case of hemichorea in which the weakness was a more prominent symptom than the movements?

The examination of the muscles and their electrical reactions did not help, as they are the same in both diseases.

Heart disease may be present in both; in the first as a cause of the paralysis, in the second as a consequence of the chorea. There seems to me only one sign which is markedly different in the two. In cerebral paralysis the reflexes are exaggerated on the affected side, and Babinsky's extensor reflex is present from a very early date; while in chorea the reflexes are either normal or merely sustained, not exaggerated. This normal reflex was present in my case, and led to my diagnosis of chorea, which was confirmed by the course of the case, which was that the movements gradually increased in frequency and violence for a fortnight, while the weakness did not alter. Then both symptoms gradually improved, and after two months' stay in the hospital the boy was discharged free from any movements and with restored strength in the affected parts.

I have brought forward this case with the object of reopening, if possible, a discussion on this interesting variety of chorea and eliciting the experiences of other members of this Society.

(*Read October the 16th*, 1903.)

Discussion.

Dr. Purves Stewart (introduced) said there are three elements in every case of chorea:—Involuntary irregular movements, muscular weakness, and ataxy on voluntary movement. Sometimes the muscular weakness swamps the movements and the result is the so-called "paralytic" chorea. In addition to the factor already alluded to, there is also invariably a mental change in the patient—an abnormal emotional state.

Why are young children immune from chorea? Why is the disease practically unknown below the age of 3 or 4 years? A possible explanation is that children of very tender years have not learned the pantomimic gestures expressive of emotion. Choreic movements, when closely studied, can usually be recognised as essentially pantomimic in character, *e.g.* grimacing, shrugging the shoulders, and other impatient gestures of the face, or limbs.

A child incapable of emotional pantomime is incapable of choreic movements. This points to chorea as an affection not primarily of the motor cortex (which produces epileptiform phenomena) but rather of the higher emotional centres.

Dr. Edmund Cautley remembered the case which Dr. Parkinson referred to and had regarded it as one of athetosis or hemichorea, due to a cerebral lesion. He objected to the term hemichorea, as applied to post-hemiplegic conditions, for there was no connection with true chorea etiologically, and the movements were different. Paralysis was always present in true chorea to a certain extent, and would be found if looked for. The extent varied in different cases and he thought that paralytic chorea in its worst aspects did not receive enough attention in solving the problem of etiology. He had seen one case in which all the limbs were completely paralysed, the muscles of the neck and back were also affected, and the child swallowed with difficulty and could not speak at all. Such a case closely simulated acute anterior poliomyelitis. Why in some instances the paralysis was so profound, while more commonly the movements were greatly exaggerated, was not clear. He suggested that it might be due to a profound toxæmia and that such cases could hardly be included under the same etiological classification as that to which Dr. D. B. Lees had recently ascribed all cases of chorea, namely, to "cerebral rheumatism." There was little or no evidence that the rheumatic poison could produce such paralysis. Were it so we should meet with paralysis in rheumatic fever.

4. A NOTE ON THE ASSOCIATION OF CERTAIN DEFORMITIES WITH AFFECTIONS OF THE THROAT IN CHILDREN.

By J. JACKSON CLARKE, M.B.

Deformities, for instance, curvature of the spine, and affections of the throat such as adenoids are of such common occurrence in children that it would be surprising if they were not frequently found associated in the same individuals and this coincidence might well be a merely accidental one. The co-existence of deformities with throat affections in many hundreds of cases that have

come to my notice in the past fifteen years awakened in my mind first a suspicion and later something like a conviction that there is more than a chance connection between the two sets of phenomena.

This impression probably has been present in the minds of many observers. In my own mind it dates from a now distant period when I was engaged in pathological work. In the course of examining the skeletal, visceral, and naso-pharyngeal changes in about fifty cadavera of children, varying in age from 2 months to 8 years, I found only one in which no signs of rickets were present (they were London children) and in that one only were adenoids absent, the roof of the naso-pharynx presenting five slight antero-posterior ridges of firm mucous membrane. Microscopical investigation, which is recorded elsewhere,* brought me to the conclusion that in rickets, besides the changes present in the bones, there is a general hyperplasia of lymphoid tissue throughout the body and that adenoids, enlarged tonsils, and simple enlargement of the cervical glands in rickets are merely evidence of this widespread affection of lymphoid tissue.

A large clinical experience has confirmed this opinion, and if I were to give the latter emphatic expression I might venture to put forward the proposition: every child who has rickets has also adenoids.

There is another class of ailing children, who differ from the rachitic: I mean children who carry an excess of uric acid in their tissues, either from erroneous feeding or as an inheritance from a gouty stock. In these curvatures of the spine, thoracic deformities, knock-knee, flat-foot, and other deformities are very common, and at the same time naso-pharyngeal hyperplasia and enlargement of cervical glands are nearly always present; and in this class also, I believe, the deformities and the lymphoid-tissue changes are effects of a common cause: the bone, cartilage, and connective tissue, like the adenoid tissue,

* In Dr. Cheadle's article in 'Allbutt's Encyclopædia of Medicine.'

being morbidly affected by the uric acid or whatever the toxic matter may be.

It may be objected that both deformity and adenoids, etc., are met with in sturdy, apparently healthy children; in such cases I would suggest that the effects have outlasted the cause.

In my own experience, if carefully looked for, evidence of rickets or of disordered digestion are to be found in such children. Another general objection to the view I have endeavoured to suggest may be urged is that the deformities of spine, chest, etc., and the slackness of muscles that leads to knock-knee and flat-foot are the effects of nasal obstruction. I am ready to admit that a state of partial asphyxia will aggravate such conditions, but the interaction of common effects of the same cause is well recognised in medicine and expressed as "vicious cycles." But a pigeon-breast may arise without there being any nasal obstruction, and the latter may be present without any deformities. This would appear to contradict my initial proposition, but it is to be explained by the varying degree in which the tissues, bone or adenoid tissue respectively, are affected by the rickets or goutiness, severally or jointly. Certain facial deformities, narrow jaws, high-arched palates, deviation of nasal septa, etc., are looked on as the effects of the disuse of the nasal passages and the production of a negative pressure in them during inspiration. This, doubtless, is the preponderant cause, but I think an altered state of nutrition in the bones is contributory. I have suspected that rickets and goutiness may have much influence in causing ocular defects by deranging the normal condition of nutrition of the sclerotic and other tissues of the eye.

Probably all this is trite to many, and I have only ventured to occupy the time of the meeting with this discursive paper because I have nowhere seen these views fully expressed. I have always carefully considered such writings from the pens of physicians as bear on these points. I have only gleaned that in children who develop

scoliosis a chlorotic condition or a "certain delicacy" is present. Perhaps some more definite statements of the case have escaped my view; if so I shall be greatly obliged to be informed where they are to be found, and I trust my friends who are physicians will excuse any lack of information betrayed in what I here put forward. Whether the above views are generally held or not it may not be out of place to attempt to point a moral, which I feel sure will meet with approval: and it is that when the surgeon has done all he can for cases of deformity or nasal obstruction the patient should be placed under the periodical supervision of the family physician for a year or two in order that the diet, hygiene, and medical treatment may be properly directed. At present it is a frequent experience of mine that a patient is sent to me for some deformity after undergoing thorough surgical treatment for nasal obstruction, and it is rare in such cases that any inquiry into the diet, etc., has been made, although there may be great scope for improvement in this direction.

Discussion.

Dr. Percy Lewis (Folkestone) said as a general practitioner it was refreshing to him to find a London surgeon giving voice to views which he had arrived at independently and published in his work on 'Spinal Curvatures.' He could not agree that adenoids are always due to rickets. He believed a large number of adenoid cases are due to mechanical causes, *i. e.* to the substitution of buccal for nasal respiration from neglected nasal catarrhs. Many cases are cured by the use of a means for preventing buccal and enforcing nasal respiration, such as wearing a wash-leather pad over the mouth.

5. CASE OF GASTRO-INTESTINAL CATARRH WITH GREAT DISTENSION.

By CHARLES MACALISTER, M.D.

The case which I am about to record is of interest owing to the fact that the severe gastro-intestinal disturbance commenced in two successive years exactly in the same week and that it was ascribed by the mother to the change of feeding of the cows which supplied the household milk. It appears that on May the 1st or thereabouts the cows in the district in which the child resided are removed from the byres, where they get dried food, and turned out into the fields. It seemed probable that this fact had something to do with the commencement of this trouble, which has on each occasion been serious, both on account of its long duration and on account of the wasting and actual danger to life which it involved.

I did not see the child during the first attack, which commenced on May the 2nd or 3rd, 1902, with severe paroxysmal pains in the stomach always associated with movements of the bowels; any food taken caused immediate action of the bowels, preceded by pain; a lienteric diarrhœa. Sometimes there was only flatus passed, but generally the motions were frothy, liquid and slimy. He had abdominal distension and anorexia and he wasted greatly. The severity of the abdominal pains rendered his parents afraid to give him food, and he was equally afraid to take it. In September he recovered after a long course of feeding on a purely animal diet rendered more digestible by the addition of pepsin with dilute hydrochloric acid. This is all I can say about this first attack the particulars of which were furnished by the child's mother, but the correctness of the dates and diets are confirmed by the prescriptions and some letters bearing upon the case which have been preserved.

Up to May the 3rd of the present year, the boy whose age was 4 years and 4 months remained perfectly well. He happened to be weighed on May the 2nd when (in a perfect state of health) he scaled 2 stone 10 lbs. On May the 4th he complained of griping pains in the stomach and I may here mention that his mother during the course of his illness (remembering that similar pains had initiated his previous illness exactly a year before almost to the day) called upon the milkman and found that the cows had been turned out to grass on May the 1st in each year. She had herself suspected that this had had to do with the trouble on the previous occasion. From this time all the old symptoms recurred, frequent griping pains in the abdomen and soft solid diarrhœa with much mucus. He at first invariably had the bowels moved after taking any food, but later the immediate relationship between eating and defæcation became less apparent.

It appears that in January he had a digestive disorder accompanied with diarrhœa and that at that time an exclusive diet of milk with some rusk was given by the doctor with the most beneficial results, and with this in his mind, the medical attendant again instituted the same diet during the first weeks of the present illness, but the little patient steadily grew worse and wasted greatly and became so distended that respiration was interfered with. The heart was displaced upwards, and the pain of the abdominal distension was added to the gripings which were evidently agonising, for the child's face and mouth became pinched and pallid, and he perspired about the lips and brows while they lasted. It was about the beginning of June when I first saw the child with Dr. Vereker and I do not think that I ever saw a more miserable little object, with wasted limbs and chest, the ribs showing up and contrasting greatly with the distended abdomen which at the umbilicus measured 25 inches. Peristaltic movements were visible and but for the fact that the bowels were moved several times daily, the motions being soft, containing undigested curds and much

mucus, one might have suspected an intestinal obstruction. The tongue was clean and covered with slimy secretion. There was no sickness. He continued in this condition throughout June and nothing seemed to modify his distress whether in the way of diet or of medicinal remedies, but the inunctions of fats stayed the wasting somewhat, for after losing 6 lbs. in May, by the end of June he had regained weight to the extent of 10 ounces.

I was away on my holiday during the greater part of June and the first week of July, and on my return found that the boy had improved a little; Dr. Carter had seen him with Dr. Vereker and strychnine had been administered with advantage. The distension was less by 2 inches. The bowels were now only moved with the aid of enemata and the addition of asafœtida to these, followed by massage, resulted in a partial deflation of the intestine.

An interesting circumstance connected with the abdominal distension was that during the night it entirely disappeared, and examination of the abdomen the first thing in the morning enabled us to conclude that no local pain or swelling and no glandular enlargement could be made out. Immediately after the first meal the distension commenced and lasted throughout the day. This indicated that there was a considerable spasmodic element in the case, but bromides, valerian, musk, opium, and other anti-spasmodics failed to influence it. About July the 9th the enemata began to be followed by the expulsion of long flakes (some of them cast-like) of coagulated mucin. It was at first thought that these were fibrinous but the microscope showed that they consisted of mucin. After this, improvement was steady and progressive, but for long there was atony of the bowels which required enemata for their relief. This was overcome by small doses of physostigmia with aloes and subsequently the exhibition of myrrh with aloes and antiseptics contributed to his recovery. The recovery of this child, although aided by these remedies, depended mainly upon his being

fed upon a purely animal diet from which however milk was excluded. The farinaceous element was represented by a small piece of rusk or well toasted bread twice in the day. This diet was commenced on July the 9th when his weight was 2 stone 4 lbs. 11 ounces; thereafter he gained about ½ lb. a week until restored to his normal weight and after a prolonged residence at the sea-side he now weighs 2 stone 11½ lbs. The abdominal distension has entirely gone and he is a picture of health.

I have brought this brief note before the Society because abdominal distention to the extent met with in this case is unusual in cases of muco-enteritis, and because it represents an instance in which the change of feeding of the cows had probably something to do with the provoking of a very severe catarrhal condition in a child presenting an idiosyncracy or some strong predisposition to be upset by grass-fed milk. (*Read October the 16th*, 1903.)

6. AN ANOMALOUS CASE OF DIPLEGIA.

By JAMES TAYLOR, M.D.

The patient is a little girl, 4 years of age, who was admitted into the hospital on October the 13th. The child had had whooping cough, measles, and chicken-pox, but otherwise was quite healthy until four weeks before, when she had an abscess on the right side of the neck, which was opened, and of which the scar is now visible. There is no history of sore throat, but soon after the abscess was opened the child became altered mentally, and became very weak, and unable to walk. It is difficult to make out from the mother, who speaks English imperfectly, what the exact condition was at the onset of the illness, but when admitted she was found to be extremely feeble, unable to stand, unable to raise

herself, and she cried, but in a very feeble way, showing considerable weakness and absence of movement of the chest. There was also occasional regurgitation of fluids through the nose, and the mental condition was one of very profound dementia. The child was dirty and unclean in her habits, and unable to feed herself, although there did not seem to be much difficulty in swallowing. In any attempt at walking at this time there was evident very great inco-ordination, and there was a tendency also to cross the legs, although there was an absence of anything like rigidity. There was very marked squinting and defect in ocular movements, although it was difficult to decide what the exact defect was on account of the child's mental condition. There did not seem to be any impairment of sensation, there was no wasting, but the knee-jerks could not be obtained. There was no change in the optic discs, and there was a high degree of myopia, and some slight alteration in the choroid, such as is usually associated with that peculiarity.

At first I thought the case was one of diphtheritic paralysis, though the profound alteration which had occurred in the mental condition was rather a difficulty in the way of such a diagnosis. I thought, however, that it was possible that the mother had not appreciated that the child's mental state had not been normal before the illness. The subsequent progress of the patient, however, has convinced me that at first there was a very marked degree of mental change, which has now almost entirely passed off. The child is now sensible, apparently understands what is said to her, is clean and proper in her habits, but she does not speak, and she does not seem to have quite the degree of intelligence one would expect in a child of four. So that if it is a case of diphtheritic paralysis, it has been associated with what I have never seen before in such a case, viz., very marked mental affection. I should, however, rather be inclined to look upon it as the effect more likely of some other toxic condition leading to alterations in the cerebral cortex, and

possibly also in the spinal cord, giving rise to a condition of what is properly, I think, described as diplegia. I think there is no doubt that many cases of diplegia in children are the result of some acute inflammation of the brain, probably associated with the presence of micro-organisms, and that the degree of both physical and mental change will depend upon the extent of the mischief and the areas of the intra-cranial contents which are most severely affected.

The child is still improving and is now able to feed herself, and on Tuesday the knee-jerk on the left side could be elicited. (*Exhibited, November the 20th*, 1903.)

Discussion.

Dr. G. E. Shuttleworth never remembered seeing a child who, immediately after diphtheria, showed any marked mental deterioration, but he recollected one case in which an attack of diphtheritic paralysis was said to be the commencement of mental impairment. He believed that in the present case there had always been a certain amount of mental inferiority, and that that might have been aggravated by the attack of diphtheria, or whatever the child had, which caused the diplegic symptoms.

Dr. Fletcher Beach regarded the case as one of cerebral diplegia rather than of diphtheritic paralysis, and he had never seen a case of mental deterioration occurring immediately after the latter condition. On the other hand, he had seen many cases of cerebral diplegia occurring with mental weakness.

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7. CASE OF MULTIPLE SUBCUTANEOUS TUBERCULOUS NODULES.

By FRANCIS JAFFREY.

The patient, a child aged 1 $\frac{5}{12}$ths years old, is suffering from multiple subcutaneous tuberculous nodules which the mother says came after the child had been vaccinated when three months old.

The child's mother appears to be healthy, and there is no history of syphilis, but her father, a brother and sister died of phthisis.

The nodules vary in extent from the size of a pea to that of a walnut. They occur in all parts of the body. When they first appeared they were hard and not painful, later the skin became red, and they softened in the centre and burst, healed slowly, and left a small scar. Constitutional treatment only had so far been adopted.

The interest in cases of this kind is owing to the fact that the relatives are convinced that the trouble is due to the vaccination, and to nothing else. It is my practice now to postpone vaccination for at least a year if there is the least suspicious nodule.

(*Exhibited November the 20th*, 1903).

Discussion.

Dr. C. W. Chapman said he remembered seeing an adult, aged about 23, with such nodules, and though she had been under various forms of treatment nothing did her any good. Finally, as a bold venture, he gave her a small dose of iodoform internally, after which they all got well, though they had been existing for years. No further nodules appeared after that treatment, and he suggested it should be tried in the present case.

Dr. Edmund Cautley regarded such cases as interesting from the point of view of prognosis. In a case he had seen, there were four similar nodules—two on the face and two elsewhere—in a baby, aged 10 months, which died ten weeks later from tuberculous meningitis. Post mortem, there were found to be four small caseating nodules in the brain, besides miliary tuberculosis in the lungs, and caseating mediastinal and mesenteric glands. In another child, aged 16 months, there were nodules of the same character on both arms, and the child recovered, there being a year later no trace of them, nor other evidence of tuberculosis.

Dr. Eric Pritchard said he saw a lady with a condition, following vaccination, very much like that of the present child. It had been going on for two years, and the abscesses opened one after another; she was never free from them, and the scar left was like that resulting from vaccination. He put her upon arsenic, and she had certainly improved; no new ones had formed. Some of the nodules had lasted three months.

Mr. Francis Jaffrey, in reply, said there was no nodule elsewhere so far as he could tell. He had sometimes used iodoform for tuberculous ulceration of the bladder with the best success. Children took iodoform very badly.

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8. IDIOGLOSSIA IN TWIN SISTERS.

By G. A. SUTHERLAND, M.D.

The patients were twin sisters, 5 years old, who were the subjects of idioglossia. There was no history in the family of a similar defect of speech, but three of the mother's sisters lisped. The patients were quite healthy, and their mental condition was apparently normal. In both children certain consonants were always altered in pronunciation in an identical manner, and words containing these consonants were similarly altered, so as to be unrecognisable. He thought the occurrence of this condition in twins was interesting, as the probability of imitation as a cause was thus excluded. He considered that there was probably a lesion in the motor centre for speech, of the nature of a delay in development, but thought the prognosis was good as the children had already improved considerably in six months, and there were no mental defects. (*Exhibited November the 20th*, 1903.)

Discussion.

Dr. Leonard Guthrie said an important point about the cases was that the fact of their being twins showed that they could not have caught that mode of expression from one another. Generally, cases of idioglossia showed a very strong family likeness to each other in regard to speech. The chief characteristic was the substitution of consonants for each other. They could pronounce the linguo-dentals clearly, but they often used them in place of other consonants, as well as substituting vowel sounds for consonants, the latter rendering the speech much more difficult to understand. In a boy, aged 8 years, under his own care, the consonants "f, l, s," were pronounced *a*, *a*. "M" was

always pronounced *âhee*, and "n" was pronounced *air*. The word "fly" would be pronounced *pâhee*, "lion" as *dâhee*. The sibilants were absent from his vocabulary. "Slipper" was rendered as *teer*, "stool" as *tooah*, "sheep" as *teeah*. The child could hiss like a snake, but could not be induced to use the "s" in its proper place. "M" which was normally rendered *âhee*, could be properly pronounced if the child were made to watch his (Dr. Guthrie's) lips, and imitate him. The child could also imitate other sounds if its attention were fixed on the speaker. But that was difficult as its mind wandered. What was at the bottom of these cases? He felt sure there was no defect in the motor mechanism of speech, because the child could be made to pronounce the letters and words when its attention was fixed. He felt certain the defect was not due to tongue-tie or adenoids, as some declared. He thought in many cases there was defective visual memory, because if the child looked away from the speaker's mouth it seemed to forget how to form the sounds. In his case if he asked the child to copy "K" on a slate, it would trace the letter carefully, but if it took its eyes off the copy and then attempted to do it, it could not. He did not think Dr. Sutherland had quite represented his, Dr. Guthrie's, meaning when he said he attributed it to some defect of hearing. He did not believe the children were deaf, but that there was a deficiency in auditory perception. He thought they could not discriminate between different sounds like other children. The condition was congenital, and he regarded it as akin to colour blindness, except that in the present case one was dealing with sounds, and not colours. The name "mind deafness" might be suggested for it. Though all cases were not alike, he thought there was a strong resemblance between them. As to prognosis, Dr. Sutherland's cases, and also his own, improved when a watch was kept upon them and they were trained. But they subsequently relapsed. He believed there would be some deficiency left in after life in all cases. He remembered a hairdresser who always used "k" instead of "s," and people used to visit his shop on purpose to hear him say "Kave, kir, or kampoo, kir?" With regard to insanity being in the family, that was so sometimes. Dr. Colman found it markedly so in his cases. In his, Dr. Guthrie's, there was nothing of the sort, but the child suffered from pseudo-hypertrophic paralysis, and a brother died of the same complaint. His sister talked as he did. The patients were not mentally deficient.

Dr. F. Parkes Weber referred to a marked case of idioglossia which he brought before the Royal Medical and Chirurgical Society * some years ago, at about the date of Dr. Guthrie's

* "Case of Dysarthria and Delay in Learning to Speak, following Cerebral Disturbance in Infancy," 'Proc. Royal Med.-Chir. Soc. London,'

paper. He concluded that in that case certainly the motor centres connected with speech were at fault, that there was defective development of these centres. Several other cases* had been published which could be best explained in that way. In his own case he thought the motor cortex of the brain was at fault, because there were no symptoms which offered any other satisfactory explanation; there was a history of an illness at the age of 2 years, which almost certainly affected the brain. The child was normal before the illness, but after it the idioglossia developed. In his case Dr. Weber had expressed a too hopeful view of the prognosis, probably because, as Dr. Guthrie had said, the speech in such cases seemed to improve rapidly at first, if a great deal of trouble were taken. But, unfortunately, there was a tendency to relapse whenever great attention was not paid, and in his case, although there might have been a little improvement since, the pronunciation still remained unsatisfactory. He thought Dr. Guthrie was illogical in saying that the fact that these children could, when they took a great deal of trouble, pronounce properly, was an argument against the cortical motor origin of the trouble. After all, with a violent effort,† people could do a good deal, which with the ordinary average effort of every-day life was quite beyond their powers.

Dr. Guthrie rejoined that he did not mean a prodigious effort, but merely that it was an imitative effort.

Dr. Henry Ashby (Manchester) said he had a bone to pick in regard to the name. Why not call such a case one of defective speech? He was exceedingly interested in such cases, and also in Dr. Sutherland's and Dr. Guthrie's explanations. He did not know whether Dr. Shuttleworth would bear him out, but a very striking fact was that in the Manchester special schools in which backward children were trained 25 per cent. of the children had defective speech, and in them substitution of an easy consonant for a difficult one was exceedingly common. He thought one factor in the condition was want of attention, which was coupled with a certain amount of brain backwardness. The normal child, long before 5 years of age, learned to speak by imitation, and he was intelligent enough to be aware of his mistakes. The backward child, whose attention could not be directed to any object for any considerable time, was apt to go

1900, vol. xi, p. 111. Dr. Weber saw the child in question (now aged twelve years) on November the 28th, 1903, after a very long interval, and found that much more improvement had taken place than he thought, the "idioglossia" having practically disappeared and only some of the old stammering remained.

* See "A Case of Arrested Development of the Speech Centre," by Dr. A. Voelcker, 'Clin. Soc. Trans.,' 1900, vol. xxxiii, p. 64.

† Dr. Weber would include effort of attention with other efforts here.

for a long while without speaking correctly. Such children were not as intelligent in any way as others of their age. In some cases, at any rate, he agreed that there must be some backwardness or fault in the motor speech centre, combined probably with a defect higher up. This centre was the most complicated piece of mechanism in the universe; it co-ordinated the muscular movements of respiration, of the vocal cords, tongue, and lips, in the production of speech. Therefore one could not be surprised that it sometimes failed.

Dr. G. E. Shuttleworth confirmed the remarks of Dr. Ashby with regard to the prevalence of indistinct speech among children in the schools specially provided for the mentally deficient. Quite one third of the number, and probably a larger proportion, were not able to speak correctly, the failure being not necessarily in the direction of a uniform substitution of certain sounds for certain other sounds, but there was a tendency to use the easier sounds when they would at all pass muster for the more difficult. There was certainly an element of laziness about it, which could be overcome by the energy instilled into them by the teacher. He was glad to say that the special teachers under the London School Board had had opportunities of instruction in the art of teaching speech somewhat after the method employed by the teachers of the deaf; and he believed that, in the cases under discussion, a course of lip-reading and speech by oral imitation would be desirable, as he thought it would help the little patients to overcome their defects. He did not quite see that the fact of these children being twins proved that the defect was not fostered by imitation. His experience was that one of twins usually took the lead, and the other followed it.

The Chairman (Dr. H. R. Hutton) (Manchester) thought that the trouble was due to defective hearing and defective observation, coupled with a general lack of energy. Many adults continually mispronounced some small word, and the refinements of sound did not seem to have ever been conquered by them. He thought the parents of many such children were not themselves exceedingly intellectual or bright, and therefore did not give the attention to the children they should. It was a difficulty to persuade the parents to screw up enough energy to correct the child's defects. The so-called colour-blindness for sound pervaded many things; it was seen in defects of speech, and also in the inability of some people to appreciate the finer sounds, such as those required in tuning stringed instruments.

9. A CASE OF INFANTILE GENERAL PARALYSIS OF THE INSANE.

By GEORGE CARPENTER, M.D.

Elizabeth C—, aged 11 $\frac{5}{12}$ths years, was admitted into the North-Eastern Hospital for Children in July, 1903. It was stated that she was in perfect health until April, 1902, when she was lost for a night, and ever since then had been "queer in the head." She has gradually been getting worse mentally ever since, being obstinate and screaming on the slightest provocation. There has been occasional frontal headache; she has slept badly and taken her food badly; she has never had any fits or seizures; ten years ago she had measles. There is no family history of insanity. Several healthy children were born to the parents. Next a child was born dead, this was followed by a living child, then the patient, and lastly a boy of eight years, who is suffering from nerve deafness of both ears.

State on admission.—She has an unusually thick covering of subcutaneous fat, and her skin is natural. Mentally she has two well-defined states. She lies awake most of the twenty-four hours, and talks incoherent nonsense to herself quite quietly and oblivious of her surroundings. Such remarks as "The goldfish are very happy," "No, Bobby is all right," and so on, can be heard by those standing by her cot. During this her face wears a placid, self-pleased expression which calls to mind the fatuous type of the general paralysis of the insane patient.

On being crossed by contradicting one of her statements, or by disturbing her physically, she bursts into an attack of loud screaming and weeping, which lasts for a few hours. She will usually answer simple questions, and is obedient to such demands as putting out the tongue until this sudden storm be evoked. She has hallucinations of pleasant objects, such as flags, ladders, and bowls of water,

which are quite common. Auditory and olfactory hallucinations have not been noticed. She continually mocks other children's cries or street cries, and imitates them well. She does not know her mother.

Her arms and legs are moved well, the former better than the latter. She can walk, and there is some slight spasticity of the lower limbs. There is a flickering tremor of the lower facial muscles when smiling or talking. The tongue is protruded normally and has an occasional tremor. There is a fine tremor of both hands, which is most marked on first gripping an object. The plantar reflexes show typical Babinski response on both sides. The elbow-jerks are active. The supinator-jerks are abnormally increased. The knee-jerks are very active and equal. There are no clonuses. So far as can be ascertained sensation is unimpaired. Her organs are normal. She cries when the desire occurs for micturition or defæcation, but has deficient power of retention, so that both evacuations are passed frequently and involuntarily. Her feet are cold and red, and sometimes blue—she has pes cavus.

She has a long face and depressed bridge of the nose. The facial muscles are generally well developed, and there is no local wasting. There are no radiating scars about the mouth. She has typical Hutchinson's teeth. Her pupils are equal. They do not react at all to light, and the accommodation cannot be tested. The fundus oculi was examined under an anæsthetic. The optic discs were pale, and there was a small patch of choroiditis in the left eye. Vision was very defective, being lost to all but light. There was no reaction to a threatening movement.

The case is of interest owing to the youth of the patient, and from the undoubted corroborative evidences of congenital syphilis associated with that condition.

(*Exhibited November the 20th*, 1903.)

Discussion.

Dr. Fletcher Beach remarked, in regard to the case of general paralysis of the insane, that he supposed Dr. Carpenter

assumed, from the presence of Hutchinson's teeth and the fact that there had been miscarriages, that it was a case of congenital syphilis, which seemed to be a very common cause of general paralysis of the insane. Some years ago he collected about 200 cases of the disease, in the majority of which syphilis was present. The general paralysis seemed to come on chiefly at the time of second dentition. Some years ago he had a case under his care which began with syphilis, afterwards had chronic meningitis, and following the latter had that peculiar type of general paralysis of the insane. He gradually lost power in his legs, and ultimately died of exhaustion. Apparently in such cases there were no delusions of grandeur, such as were met with in the adult type, but simply screaming and emotional disturbances.

Dr. James Taylor, speaking in reference to the case of general paralysis, said he thought such cases in children did not fall into quite the same clinical category as the adult cases, and they had not the same general history. Dr. Carpenter's case was an illustration of an unusual form in his, Dr. Taylor's experience. Those he had seen had commenced with gradual dementia, and had associated fits, tremors, inactive pupils, and excessive or absent knee-jerks. In the majority there was of course the very definite association of congenital syphilis, as in Dr. Carpenter's case. In the cases published by Dr. Mott in the 'Archives of Neurology,' he did not think there was a single case in which congenital syphilis could be excluded. In the present case that evidence was undoubted, and if one could divide the clinical history of the disease in children, Dr. Carpenter's was in the third stage. The cases in children were definitely related to those in adults, in virtue more particularly of the physical conditions, and not so much on account of the similarity of mental conditions; the latter in children was simply dementia, rarely if ever delusions of grandeur. It was an illustration of congenital syphilis producing the disease, and he believed congenital syphilis did produce certain definite conditions in children, and also certain indefinite ones. Amongst the more definite ones were the cases of general paralysis of the insane, and, congenital tabes dorsalis,—not to be confounded with Friedreich's ataxy.

10. PREMATURE GREYNESS OF THE HAIR OF THE SCALP IN A CHILD 11 YEARS OLD.

By GEORGE CARPENTER, M.D.

Laura M—, aged 11 years, attended the Out-patient Department of the North-Eastern Hospital for Children in October, 1903, for greyness of the hair of the head dating from the previous January. For the past nine months she had suffered from slight attacks of headache, which were general, came on once a month, and lasted for two or three days. For the past five years she had been maltreated and several times frightened by a brutal father. There is no history of premature greyness in any member of the family.

On examination she is a healthy-looking, well-nourished child. The altered condition of her hair is the most striking feature of her condition. Originally of a light brown colour, it is now largely interspersed with grey or even white hairs, so as to resemble the normal hair of a woman of 55 years. On close examination one finds that the grey appearance is fallacious, as most of the hair is definitely either pure white or blonde and the admixture seems grey. The superficially exposed hair is blonde, whilst the white is confined to the sheltered part. The white hair appears everywhere. It is especially marked on the front and sides, and less so behind. There is a broad, ill-defined band passing down the middle, and this marks off a bundle of hair which is white for the proximal two thirds and blonde for a distal exposed one third. She is mentally very intelligent. The heart's action is readily quickened. Her extremities are blue and cold. All her organs are healthy. The iris is of a light grey colour. The lower part of the abdomen is mottled with pigmentation not unlike pityriasis versicolor.

(*Exhibited November the 20th*, 1903.)

II. THREE CASES OF SARCOMA IN CHILDREN.

By DOUGLAS DREW, B.S.

The following cases are of interest, inasmuch as sarcomata are not very frequently met with among children, and are particularly uncommon in young babies; and the results of operations for their removal are generally so disappointing that it appears to be worth while to record cases in which success attends our efforts to eradicate the disease.

Case 1.—*Round and spindle-cell sarcoma of the muscles of the forearm.*

L. B—, aged 6½ years.

The tumour, which had been present four and a half months, formed a well-marked swelling about the middle of the back of the right forearm. It appeared to be situated deeply among the muscles to which it was fixed, but it was not adherent to the bone. It was soft, and presented a well-marked rounded edge. There was no enlargement of the axillary glands or evidence of other growths.

The tumour was removed on April the 28th, 1903, through a longitudinal incision made over it. It was of pink colour, lobulated and encapsuled, it lay beneath the extensor muscles, and was partially embedded in the supinator brevis. At the lower part there was evidence of infiltration into the substance of the muscles. The tumour was removed, together with some of the muscular tissue.

Under the microscope the growth proved to be a round and spindle-cell sarcoma.

Remarks.—The malignancy of the tumour was so marked that it appeared that little good would result from its removal, and that evidence of recurrence would rapidly appear. However, six months have elapsed since the operation, and up to the present the child is free from disease. It is too early yet to think that the patient is

cured, but should so fortunate a result accrue, I will bring the child before the Society at some future date.

Case 2.—*Adeno-myxo-sarcoma of the left testis.**

A. J—, aged 16 months.

History.—Enlargement of the scrotum had been noticed three months. This proved to be a hydrocele, and was tapped, but no enlargement of the testis was noticed. A month later it was tapped again; very little fluid escaped, and the testis was found to be enlarged. The tumour caused a general enlargement of the testis, the organ being about equal in size to that of a child of about 10 years. It was firm and not tender, and there was no thickening of the cord or vas deferens. The testis was removed on April the 15th, 1902, the cord being drawn down and ligatured as high as possible without opening the inguinal canal.

Three and a half months later some slight nodular thickening was noticed in the tissues of the scrotum. This was removed with a surrounding layer of tissue, and on section proved to be a soft, pinkish growth measuring a quarter of an inch in diameter. There was no evidence of thickening in the inguinal canal.

Sixteen months have now elapsed since the second operation, and the child is free from recurrence.

The microscopic report on this specimen is interesting, in that it shows that the tumour was largely composed of adenomatous growth with a sarcomatous stroma. The freedom from recurrence is probably due to the less malignant nature of this kind of growth.

Microscopic report.—A section of this specimen is composed of numerous glandular spaces which have become filled with intracystic papillomatous growth. These ingrowths exhibit advanced mucoid degeneration of their stroma, and there is similar degeneration in other parts of the section. It is probably an adeno-sarcomatous tumour,

* For sequel see "Secondary Angiosarcoma of the Lung in a Child aged Three Years," No. 42, p. 120 in this volume.

because of the cellular nature of the stroma between the cystic spaces.

Case 3. *Round-cell sarcoma of the upper jaw.*—F.M—, aged 3 months, was brought to the hospital with a rounded thickening of the upper alveolar border in the middle line. The gum appeared normal except on its anterior aspect, where it was of a dull red colour and soft to the touch, and there was a small ulcer resulting, according to the mother's statement, from a puncture made by her doctor, from which some fluid escaped. The growth, which was limited to the alveolar border, was freely gouged away and the cavity plugged. It healed rapidly, leaving the left central incisor teeth presenting; these, as they were loose, were removed.

At the end of six weeks distinct thickening of the bone could be felt around the left nostril. The growth was exposed by dividing the lip and carrying the incision around the nostril and upwards along the side of the nose. The tumour appeared to be quite limited to this part of the bone, so the jaw was partially removed, leaving the orbital plate and the posterior portion of the bone, which appeared healthy. On dividing the bone, the antrum was found to be full of soft pinkish friable growth, but this was not attached to the mucous membrane; the latter, however, was carefully dissected away.

The infant bore the operation well, but collapsed and died suddenly shortly afterwards.

The microscopic report on a section of the original tumour is interesting. It is as follows: "This is a very peculiar growth of the jaw, more especially as it is stated to come from a child. It is distinctly alveolated like a carcinoma, and a large number of the cells have an epithelial appearance; but in the centre of many alveoli there are collections of smaller round cells such as would be called sarcomatous. I believe the tumour is an example of a sarcoma arising in the dental follicle, and I would suggest that the peculiar appearance described above was

probably produced by the sarcomatous tissue invading the epithelial tissue of the enamel germ of the permanent incisor tooth." (*Read November the 20th*, 1903.)

12. CASE OF CONGENITAL HEART DISEASE, WITH DYSPHAGIA.*

By ERIC PRITCHARD.

An infant of three months was admitted as an out-patient under Dr. Rolleston, at the Victoria Hospital for Children, for difficulty in swallowing. The mother had suffered from ill-health and shortness of breath before the infant was born, and both paternal and maternal grandfathers had died of angina pectoris. The infant was more or less cyanosed from birth, and congenital heart-disease was stated to have been diagnosed soon after birth. The prepuce was very long, and the orifice completely occluded, so that circumcision was necessitated on the day after birth. This operation was performed without an anæsthetic. The dysphagia was noticed soon after birth, and the infant, who was breast-fed, appeared unable to suck or swallow properly. He was ultimately fed with a spoon, and the milk was drawn off with a breast-pump.

The difficulty in swallowing appears to be due to inco-ordination of the muscles of deglutition, rather than to organic obstruction—the movements of the tongue are clumsy and purposeless.

There is a loud systolic murmur audible over the whole cardiac area, but loudest along the left border of the sternum.

The cyanosis and dysphagia have improved, but the infant is rapidly sinking into a marasmic condition.

(*Exhibited November the 20th*, 1903.)

Discussion.

Dr. G. A. Sutherland thought cases of difficulty in swallowing in young infants were not uncommon, and he believed it was

* For sequel see No. 30, p. 93 of the present volume.

due chiefly to inco-ordination of the muscles of the throat. He had examined for any mechanical difficulty, such as an enlarged thymus, enlarged glands, and so on, but he had not discovered any such condition in the majority of cases.

13. CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By EDMUND CAUTLEY, M.D.

The case, which I am recording, presented many unusual features. It illustrates the difficulties sometimes found in making a diagnosis and in deciding on the appropriate treatment. It also shows that the affection may be easily overlooked and why such a mistake may arise. That the affection is not uncommon is evident from the number of cases recorded in recent years and the number which come under the notice of individual observers. In a paper read before the Royal Medical and Chirurgical Society in November, 1898, I ventured to state that, although I only recorded twenty cases, including my own and others published, in my opinion "the condition is not so rare as might be supposed" and that it is distinctly the cause of that symptomatic disease, marasmus, in some babies. In a joint paper with Mr. Clinton T. Dent on the affection and its treatment by pyloroplasty, read four years later before the same Society in December, 1902, it was mentioned that the total number of recorded cases exceeded fifty. In other words more than thirty fresh cases had been recorded in four years. Since then three more cases have come under my care, and altogether I have seen ten cases in less than seven years. It is clear that the affection cannot be very rare and that the more generally attention is attracted to it, the more frequently it will be recognised. Such recognition is a most important matter seeing that operative treatment has proved

curative, while in the absence of such treatment the prognosis is almost hopeless.

Douglas G—, born June the 22nd, 1903, was admitted into the Metropolitan Hospital on September the 22nd and died on October the 3rd, aged 14 weeks.

He was the first child of healthy parents, and, though supposed to have been born four or five weeks prematurely, was said to have been a very large and fine baby. He was nursed for six weeks, then fed on Allen & Hanbury's No. 1 food, and after that on humanised milk up to August the 11th, when he was brought to my Out-patient Department during my absence from town for my summer holiday.

At that time he weighed 6 lbs. 11 oz. and was said to be suffering from vomiting and wasting. He was ordered a mixture of cow's milk and barley-water. On August the 18th he weighed the same, had vomited occasionally, and had rather frequent stools. On August the 25th he had gained 1 oz. On the 28th he had lost 1 oz., had developed thrush, and still vomited occasionally. On September the 5th he had lost 2 oz., weighing 6 lbs. 9 oz. During the next week he gained 7 oz. and a further 6 oz. in the following week; a noteworthy gain. On September the 22nd I saw him for the first time and after careful examination admitted him as a case of congenital hypertrophic stenosis of the pylorus. He then weighed 6 lbs. 14 oz.

The important points in the history and course of the disease were as follows: According to the mother the vomiting began at the age of 3 weeks, had steadily persisted, and had become more frequent. It did not possess the usual characteristic that several feeds might be kept down and then a large quantity of food would be brought up. The bowels had been costive and the stools small. The child had steadily wasted except from September the 1st to the 15th, when he gained 13 oz.

On physical examination it was noted that he was very wasted, the fontanelle was depressed, the tongue clean,

and the breath sweet. The abdomen presented a fulness in the epigastric region and the outline of the stomach was ill defined. The stomach was hyper-resonant, a little dilated, and exhibited visible peristalsis passing in a wave from the fundus to the pylorus. A movable lump, the size of a filbert, could be felt in the region of the pylorus. He was put on a diet of equal parts of peptonised milk and water every two hours.

September 23rd. Vomited four times, immediately after food, up to 4 a.m. The amount vomited was small and consisted simply of the food.

September 24th. No more vomiting. Takes well. Bowels open, stools small. No loss of weight.

September 25th. Vomited twice during the night. Passed a large yellowish stool this morning, containing a few curds.

September 26th. Loss of 3 oz. Vomited once, rather a large amount. Bowels acted twice.

September 28th. Gain of 6 oz. No more vomiting.

September 29th. Passed four stools during the night, green, some curds.

September 30th. Loss of 6 oz. Has vomited once slightly. Stools green, no curds.

October 1st. Passed seven stools yesterday, containing little fæcal matter; only one stool to-day. Has vomited once.

October 2nd. Loss of 7 oz. Passed four stools during the day. Vomited once, about an ounce and a half, while the stomach was being examined. Pylorus still felt. No increased dilatation of stomach.

October 3rd. Passed two stools; vomited once. Died from exhaustion, due to marasmus, in the evening.

The temperature ranged between 97° F. and 99·6° F. throughout.

On post-mortem examination, eighteen hours after death, nothing abnormal was found except the condition of the stomach and pylorus. On opening the abdomen the distended stomach was seen to occupy the left hypochon-

drium and the epigastrium down to the level of the umbilicus. The pylorus formed a definite cylindrical or sausage-shaped tumour about an inch long. The stomach contained an ounce and a half of dirty fluid, a little altered blood, and a moderate amount of gas. It was not found possible to squeeze any of the contents of the stomach through the pylorus, although a probe could be passed through. On longitudinal section the pylorus showed the changes usually seen in this affection, namely, a marked hypertrophy of the circular muscular fibres. A good illustration of this was published in the 'Lancet,' p. 167, December the 20th, 1902. The small and large intestines contained a considerable quantity of normal yellow fæces.

First, I wish to draw attention to the difficulties in making a diagnosis in this case. Previous experience had induced me to form the opinion that as a rule the diagnosis is fairly obvious in a case under observation for a few days. In a typical instance we find progressive wasting; vomiting, unaccompanied by nausea, and characteristic of pyloric obstruction; absence of bile from the vomit; constipation; clean tongue; sweeth breath; a dilated stomach; visible peristalsis; and a palpable pylorus.

Palpation of the pylorus is difficult on account of its deep position, and one is apt to hesitate in stating with absolute confidence that it can be felt. I have felt it in the last five cases I have seen. Other tumours in this situation at an early age are very rare. An enlarged gland, if present, is not as definite and does not give rise to similar symptoms. Gastritis can generally be excluded on account of the clean tongue and sweeth breath.

In the present case the vomiting was certainly not characteristic. Although it had persisted for a long time and had become more frequent, there was no history of a few feeds being retained and then all brought up at once. During the twelve days the child was under observation in hospital the vomiting was not marked and had not the typical characters. The bowels were stated by the mother

to be costive. While under observation they acted fairly often and contained a considerable quantity of fæcal matter. On one occasion, eight days before death, a large, almost normal stool was passed, conclusive evidence against very serious obstruction.

Dilatation of the stomach was not very marked and did not increase while the child was under observation. Peristaltic movements in the stomach were noted on one occasion, but were never present to the marked extent seen in some cases.

Another unusual feature was the extraordinary gain of weight which took place twice in the course of the illness, instead of the usual steady progressive loss. During one fortnight, while attending as an out-patient, he gained 13 ounces. During two days, while an in-patient, he suddenly put on 6 ounces. If a child can gain weight like this, there does not seem to be any urgent need of operation. Nor does there appear to be any reason why such improvement in nutrition should not continue. The affection was diagnosed from the wasting, vomiting, dilated stomach, and the palpable pylorus. On the absence of characteristic vomiting, on the presence of the considerable amount of fæcal matter in the stools, and on the two periods of gain in weight, I decided that the obstruction was only partial, that food could get through, and at times in sufficient quantity for perfect nutrition, and that under such conditions the operation might be postponed. The child was very wasted, and I hoped it might be possible to get it into a better state of nutrition or that it might be cured without operative interference. Even when he started to lose ground rapidly food was still passing through the pylorus, the vomiting was not marked, and the general indications for operation did not seem particularly urgent, looking at them from the point of view of relieving an obstruction.

Now note the peculiarity of the conditions found at the autopsy. The pylorus would not permit the passage of fluid even. No doubt this was due to post-mortem con-

traction. Note, too, that the intestines contained a considerable quantity of normal fæces. Clearly food had passed through the pylorus within a few hours of death, and the degree of stenosis found at the autopsy is no measure of the obstruction present during life. The absence of characteristic vomiting during life is suggestive of the absence of spasm during life, the obstruction being due to a mere hypertrophy with secondary contracture of the hypertrophied muscle.

The child's life might have been saved by operation. Four out of my last five cases have been operated on by Mr. Clinton T. Dent, and life has been saved. In the present case no operation was done, and the child died. He died from marasmus induced by starvation, from insufficient food passing through the pylorus. At the time he came under my care it is more than probable that he would have died from the marasmus, in spite of surgical treatment, though the latter would have undoubtedly given him a better chance.

From this case it is clear that the affection can exist apart from typical symptoms; that an apparently mild case can induce severe wasting; and that operative treatment is indicated, although food can pass through the pylorus.

There is little doubt that the extent of the condition may vary in different cases, and that both the diagnosis and the decision as to the necessity for operation may be matters of very great difficulty. I believe that mild cases may exist; that the symptoms may not be of much moment, and may be limited to dyspepsia and occasional attacks of vomiting; and that such cases may terminate in later life from dilatation of the stomach, and hypertrophic stenosis of the pylorus.

Such cases have been recorded at 5 years (Hezekiah Beardsley); at 6 years (Sonnenburg); and at 11 years (Hansy). Probably hypertrophic stenosis of the pylorus in the adult is simply due to the persistence and exaggeration of a mild type of the affection in infancy.

(*Read November the 20th*, 1903.)

14. ON CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By C. T. DENT, M.C.

The number of cases of congenital hypertrophic stenosis of the pylorus that have been operated on in infancy and published* is still so small that it seems desirable to set down some recent clinical experience on the subject, more particularly for the reason that the cases illustrate in their sequel many of the difficulties likely to be experienced. Highly typical cases of the affection may be expected to run a fairly straightforward course, but a fuller knowledge of the affection and the best method of treating it can only be gained by the accumulation of clinical observations.

The first case was that of a patient born on June the 4th, 1903. The mother's age was 33, and the child was her first. The labour was reported to have been a bad one, and the child was delivered with forceps. It weighed at birth about 7 lbs. From the beginning of its life it was fed by hand. Ten or twelve days after birth the symptoms commenced. It was noticed that the child was soon satisfied. The characteristic vomiting then commenced, *i. e.* three or four feeds might be kept down, and then the accumulated stomach contents were violently ejected. Various kinds of foods and medicines were tried, but without effect. Gastric lavage was not employed. When I first saw the child with Dr. Cautley on July the 23rd, the symptoms pointing to the condition, which had been suspected for some little time, of congenital stenosis were so pronounced that it was decided to operate at once.

At the time of operation the child weighed an ounce or two over 5 lbs., having therefore lost about 2 lbs. since its birth. It was exceedingly small and wasted. Gastric

* In a paper in vol. lxxxvi of the 'Med.-Chir. Trans.' will be found a *résumé* of twenty cases.

peristalsis was occasionally evident. A thickened pylorus had been felt, but when I saw the child I could not make certain that there was any thickening, as the abdomen was a little distended. Even slight distension will prevent the pylorus from being felt. It is not that the thin and supple edge of the liver obscures the pylorus, for in these cases, usually, the pylorus will be found rather lower and rather nearer the middle line than might be expected. The child was fifty-one days old at the time of operation.

The abdominal wall was excessively thin, and the peritoneum almost burst open when exposed. The pylorus was at once felt and drawn up into the wound. It presented a very characteristic and strongly marked instance of congenital hypertrophic stenosis. The thickening involved the pyloric antrum partially, but extended also some way down over the commencement of the duodenum. The thickening was nearly an inch in length, and the tumour had the white appearance that is usually seen in these cases. The stomach was not dilated. Pyloroplasty was performed. A longitudinal incision about an inch and a quarter in length was made through the thickened tissues from the stomach down to the duodenum. The hypertrophied muscle was about half an inch in depth, and gave the characteristic creaking sensation as it was divided. There was marked absence of submucous thickening, and the mucous membrane was very pale. As usual, directly the opening was made into the stomach and a little gas had escaped, the remaining steps of the operation became easier. At the upper and lower extremities of the incision the mucous membrane was united to the stomach and the duodenum respectively. This was done in order to prevent crumpling up of the mucous membrane and consequent obstruction when the pyloroplasty was completed. The central, the most important stitch, was closed first. I have noticed in other cases that there may be considerable drag on this stitch at first owing to the action of the longitudinal muscular fibres of the stomach. If, however, this stitch is closed up rather slowly, the drag can be over-

come without any risk of the stitch cutting through. When once closed there is no fear of it giving way. In some instances it may be found advantageous to close the lateral sutures before the central. The best form for the central stitch is a Halsted's suture. The remaining sutures came together satisfactorily and without any difficulty, notwithstanding the marked hypertrophy of the circular fibres. No food was injected into the duodenum at the time of operation. I have always had a feed ready to hand, but I have never actually yet injected it while operating. The proceeding would entail delay, and rapidity in these cases is of the first consequence. Five sutures in all were put in, a quite sufficient number to convert a longitudinal into a transverse wound and to close up the opening completely. An omental graft could easily have been added, but I refrained from doing this, as it appears to me to be unnecessary and likely to lead to after-trouble. The operation from the commencement to the time that the child was replaced in bed occupied twenty-five minutes. The anæsthetic—a highly important factor in these cases—was admirably given by Mr. W. F. Fedden.

Rectal feeding was employed for the first twenty-four hours, small quantities of hot water being given by the mouth. I think there is no objection in these cases if it is thought necessary, and if the rectal feeds are not well retained, to giving food by the mouth after twelve hours or so; but some blood is generally vomited up after the operation, and so long as there is blood in the stomach it is unlikely that food will be retained. Next day the child took its food—peptonised milk and water—by the mouth fairly well. Two days later the vomiting had practically ceased, and it was evident that the passage through the pylorus was thoroughly patent. The temperature, however, was rather high, though the wound gave no trouble. The child was for many days kept back by constant diarrhœa due to enteritis. The stools were green, as they had been throughout the child's life, and much of the milk was found, undigested, in the stools.

The after-treatment was mainly directed to improve the enteritis. This trouble gradually improved, but the child's general condition mended but slowly. In this instance I had to deal with a rather delicate child in somewhat poor general condition, and operated on rather late. I have little doubt that if the operation had been performed earlier the after-treatment would have given rise to less anxiety. My own experience of these cases leads me to the strong belief that the operation is decidedly indicated when the weight continues to diminish under medical treatment or lavage; and this view is strongly emphasised by the information I have been able to collect, and by a study of the literature of the subject. It is possible that with such treatment the urgency of the symptoms may subside, but the mechanical condition of the pylorus still remains—a point to which I will return,—and there is a great and a constantly increasing risk of gastric dilatation, as well as of grave intestinal changes.

The child left the hospital on August the 27th. The parents lived in a very healthy place, and it was thought that the fresh air and surroundings, other than hospital, might improve the general health.

On September the 22nd, however, as the accounts of its progress were very unfavourable, it was readmitted. The child was then in a very emaciated state, and weighed only 5 lbs. 12 oz., *i. e.* it had lost 14 oz. in weight since it had left the hospital. There had been some vomiting, but this had not the old character, the stomach contents not being forcibly ejected. The belly was much distended, and the coils of intestine could be plainly seen. There seemed little or no intestinal digestion, and a great deal of fermentation in the intestinal tract. The stools were very colourless. The vomiting soon ceased under altered conditions of diet and skilled nursing. In this case I found humanised milk answered well. Raw meat juice (cold extract) and cream were also added to the diet. Under this regimen the child soon began to improve, and as the enteritis subsided it began to put on weight fairly

rapidly. It was not, however, till the child was nineteen weeks old that its weight was equal to that at the time of birth. It then began to improve much more rapidly, and at the time of writing looks well and happy, and weighs 10 lbs.

The case illustrates well the necessity for early operation in these patients. Delay will always, I believe, increase the difficulties and the anxieties of the after-treatment.

A consideration of the conditions will explain easily enough the sources of the trouble, and the slow improvement when operation is performed in a late stage. However pronounced the degree of hypertrophic stenosis, we may assume that some fluid passes through the pyloric opening. The copious vomit, however, furnishes clear evidence that the amount that escapes from the stomach into the duodenum may be very small. Now physiologists hold that the stimulus to the secretion of the pancreatic juice consists in the introduction into the duodenum of the acid contents of the stomach. The bile is probably excreted continuously. At the same time it seems reasonable to suppose that if the pancreatic juice is diminished in quantity the output of bile is often less. Two conditions then may obtain :—(1) The secretion of the pancreatic juice may be excited by the tardy introduction of the stomach contents into the duodenum, in which case there will be, as it were, an excess of pancreatic fluid in the intestine—more than is sufficient to deal with the minute quantities of food that pass on from the stomach. (2) If the reaction of the gastric contents long retained in the stomach does not preserve its acidity, then, when the food is passed on at an unnaturally late period after it has entered the stomach, the stimulus to the pancreatic secretion will no longer be furnished. The digestion of the fatty elements, therefore, must be gravely interfered with. It becomes, then, very important to ascertain in each case what the reaction of the stomach contents is when the vomit consists of accumulated feeds. Hitherto it does not appear that sufficient attention has been paid to this point. If the gastric contents from long retention

become neutral or alkaline, then the principal stimulus to the secretion of pancreatic juice is abolished, and any portion of the food that has passed through the obstructed pylorus can only be so imperfectly digested in the intestinal tract as to have no nutritive value.

The opinion is now held by physiologists, in marked contra-distinction to the old teaching, that no absorption of water takes place in the stomach itself. Very small quantities of proteid material are taken up. The progress of these children supports this view. In marked cases the patients die about the twelfth week after birth; and they die of starvation. The vomit is so copious that it is evident that very little, if any, of the water has been absorbed from the stomach; at the same time, sufficient absorption of nutritive matter might almost be taken up from the stomach alone to keep the children alive, say, for the few weeks for which they live, if unrelieved.

Of equal importance are the changes that must be brought about in the intestinal tract, particularly in the small intestine. If no food at all gets into the intestine, a point is reached, before actual death occurs from starvation, when recovery is impossible. The epithelial cells that have a selective power in the absorption of food from the intestine become atrophied, and when this stage is reached the introduction of food is useless, for it cannot be absorbed. Not only is nutrition impossible, but material introduced becomes an irritant when it ceases to be a food. In acute cases of hypertrophic stenosis the process of death is due to tolerably rapid starvation. Gradually an atrophic condition of the intestine is brought about, and these changes furnish a very simple and physiological explanation of the principal difficulty that is met with in the after treatment.

The consideration, then, of the changes that must result from the form of obstruction under consideration in the case of a young child, emphasises strongly the necessity for early operation. The importance of giving mechanical relief to a mechanical disorder is still more marked in

the case of infants than in adults, for the intestinal changes will probably go on more rapidly in the case of the very young. Not many years have elapsed since the tendency to undue delay in operating in most forms of intestinal obstruction, save in strangulated external hernia, was unfortunately only too marked. The results were disastrous. Surgeons were not called in until the condition was almost hopeless. Palliative operations—if any operations at all were performed—were the rule, and the want of success was so great when these half-measures were adopted, or when more radical measures, in rare instances, were employed, that acute or subacute intestinal obstruction came to be looked upon almost as a hopeless condition. Matters improved as soon as the necessity for early operation was recognised. The same considerations apply in the case of this form of pyloric obstruction. After all, the condition is but a form of intestinal obstruction, and it may be nearly complete. It will not get well of itself, if at all marked. It will lead to early death in many instances, and even if ameliorated by medical measures, by gastric lavage and so forth, it is practically certain that the condition will persist.

It must be kept in mind that improvement does not follow always—indeed, seldom follows—immediately on operation. Rectal feeding may be necessary for some hours after operation, and the life of a greatly reduced and emaciated child, that has been suffering from constant vomiting, is very precarious when it depends for its nutrition on what it can absorb by the rectum.

Although it is, perhaps, premature to assert as an established fact that the condition, if present in a mild form, may persist through life, yet a sufficient number of cases are now on record to render it practically certain that such is the case.

It has been found necessary to operate on cases of hypertrophic stenosis of the pylorus at all ages, and the probability that most of these were really of congenital origin amounts almost to a certainty. Some authors—

Hemmeter, *e. g.**—hold the view that these cases not occurring in infancy are the result merely of chronic gastritis. Without denying altogether that this may be the case, I feel quite sure that in most of the cases referred to in Hemmeter's book the chronic gastritis was a secondary symptom, and that many at least of the instances referred to in his remarks on non-malignant stenosis of the pylorus were essentially cases of a milder type of congenital hypertrophic stenosis. Plate x in Hemmeter's work shows a section of part of the stomach and duodenum from a case described as "Hypertrophic Stenosis of the Pylorus from Chronic (stenosing) Gastritis." The section appears from the text to be from an adult stomach. The resemblance to the condition that may be found in infants only ten or twelve weeks old is very striking. Reference to the illustrations that will be found in vol. lxxxvi of the 'Med.-Chir. Trans.,' accompanying the paper already quoted, will show the similarity at once. Proportionately, the amount of muscular tissue is the same. The chief difference lies in the amount of thickening in the submucous layer in Hemmeter's figured case. Dr. John Thomson has seen similar thickening in cases of congenital hypertrophic stenosis. To my mind the condition figured in Hemmeter's book may well have existed from birth. If this be so, the arguments in favour of early operation become exceedingly strong. The lifelong troubles that some of these patients have to endure might, there is every reason to think, have been obviated by early operation. At the same time I fully allow that as yet we do not know enough of the after-history of cases operated on in infancy to justify us in assuming positively that a permanent cure can be effected. The first recorded instance that I have been able to find of an operation done for congenital hypertrophic stenosis in infancy dates only from four years ago.

That the operation at a very early age is more difficult must not deter us from operating. The youngest patient

* 'Diseases of the Stomach,' by John C. Hemmeter, 2nd edit., p. 617.

on whom I have operated so far was thirty-four days old. In this case there was strongly marked hypertrophy of the circular muscles about the pylorus, but the child was still in good condition. The vomiting in this fourth case did not cease entirely for some days after operation, but then the child began to improve very rapidly. It is worthy of note that this child was exceedingly fortunate in being under the care of a most admirable nurse. The nursing of these patients after operation is one that demands the utmost skill, judgment, and care. It is too early yet to speak positively about this patient, for the operation was only performed on October the 23rd last, but at the time of writing the child has lost all its grave symptoms, is gaining weight, and seems perfectly well and happy. Still, even in this case, in which there was prolonged constipation before operation, there are slight digestive troubles which require watching.

I am still of the opinion, from the four cases that I have had, that pyloroplasty is the best operation that can be performed. The difficulties of this operation appear to me to be quite theoretical, and the experience at any rate of four cases, all of which have recovered, appears to me to give a flat denial to the views of those authors—and they are by no means few in number—who argue that the operation is altogether impracticable.

Elsewhere I have urged that dilatation of the pylorus or pyloroplasty are the best operations, and I am still of the same opinion, with a decided preference for the latter method. Gastro-enterostomy—an operation of immense value that has been rather indiscriminately employed of late—is a proceeding so much in favour at present that it would be strange if it had not been advocated as a treatment in congenital hypertrophic stenosis. Gastro-enterostomy has, indeed, been spoken of as an ideal operation for the condition. The epithet seems rather strained when we reflect that it leaves untouched the essential cause of the trouble, a cause too, that, as I have furnished evidence for believing, will persist. To my mind gastro-

enterostomy, as applied to these patients, is but a palliative makeshift. Bearing in mind the nature of the affection, it is surely better and certainly more scientific to deal with the part really at fault. That the condition of the pylorus can be remedied directly and efficiently there are now recorded cases enough to prove.

(*Read November the* 20*th*, 1903.)

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15. A CASE OF HYPERTROPHIC STENOSIS OF THE PYLORUS.

By ALEXANDER MORISON, M.D.

Dr. Morison showed the stomach of a child, aged 2 months, in whom pyloric hypertrophy had been diagnosed and who was admitted to the wards in the Children's Hospital, Paddington Green, for operation. While under observation, the patient vomited very little, and only occasionally exhibited gastric peristalsis. The child returned to its home for a few days, instructions having been given that it should be brought back if vomiting returned. Five days later it was readmitted, collapsed, and with a temperature of 90° F., and although it rallied somewhat, it died two days afterwards. The stomach, inflated with air, only required the cardiac end to be ligatured to remain inflated. The pylorus was large, long, and hard. The great hypertrophy was well shown by comparison with the stomach from a child seven months older than that which was the subject of hypertrophy, and than which the affected pylorus was much larger and harder in all respects. Dr. Morison dwelt on the necessity for operation in such cases, even when the condition was merely suspected to exist.

(*Exhibited November the* 20*th*, 1903.)

Discussion.

Mr. Howell Evans said he had met with a case in which there was associated with congenital stenosis a marked degree of spasm. This example suggested that an anæsthetic might sometimes be of use in these cases. The instance he referred to was one of twins, the child weighing 5 to 6 lbs. at birth, and it thrived moderately for about a week. Then it began to show evidence of dilatation of the stomach. The vomiting was just the same as one would expect to find in an adult, with stenosis of the pylorus. An anæsthetic was administered, but the child seemed very poorly circumstanced, even for a five or ten minutes' surgical operation. He made his first incision through the skin, being ready almost at the next to advance forward, his object being to find the pylorus, and by invaginating his finger through the stomach to ascertain the condition of the pyloric orifice. If it seemed distinctly constricted, he would have done a pyloroplasty; if not, he would have left it alone. But the child became so collapsed that he had to abandon the operation. The infant subsequently was fed upon very small quantities at very frequent intervals—about two tablespoonfuls every half hour—for a considerable time. It is still alive, the symptoms of stenosis of the pylorus have disappeared, and the progress of the infant has been marked.

Dr. G. B. Turtle (introduced) said that during the last twelve months he had seen five cases such as those described, all of which were operated upon by Loretta's method, and all recovered. He asked whether Mr. Dent had any difficulty in sewing up the thickened pylorus, and whether there was any leakage afterwards. Also, did Mr. Dent think there was more spasm than hypertrophy? An interesting point was that two of the cases returned later with diarrhœa of a very troublesome nature. They were admitted into hospital, but the diarrhœa was very difficult to stop. He thought that might be due to the pylorus being so over-stretched that it was unable to retain the food in the stomach long enough for digestion to take place, and that therefore undigested food passed on to the intestines.

Mr. Clinton Dent, in reply, said there was no difficulty in sewing up the pylorus and there had been no leakage in any of these cases. He had already in a paper read before the Royal Medical and Chirurgical Society stated that in his opinion there was not very much to choose between the operations of pyloroplasty and forcible dilatation; but additional experience led him to consider that the advantages of the former operation over the latter were decided. At any rate in all his four cases he was convinced that pyloroplasty was best suited to remedy the condition found.

16. AN ABDOMINAL CASE FOR DIAGNOSIS.

By ALEXANDER MORISON, M.D.

Dr. Morison said the patient was a boy, aged 9 years, who came to the Out-patient Department of the Paddington Green Hospital with œdema of the face and genitals, and of the feet. He was taken into the hospital, and his urine was found to be practically solid with albumin on boiling, the quantity passed having diminished to 11 to 14 ounces of specific gravity 1028. The œdema increased, the legs and abdomen became much swollen, free fluid appeared in the abdomen and the loins pitted on pressure. The heart was normal, but some œdema of the pulmonary bases was present. There was very little deviation of the temperature from the normal. When he had been in hospital nearly a month œdema of the genitals became so extensive as to cause retention. Puncture of the genital œdema was performed and 20 ounces of urine were found in the bladder. Afterwards he passed fairly large quantities, and the specific gravity fell to 1010, with a cloud of albumin. There had never been blood in the urine, only hyaline casts. The hospital having been closed, the child was sent to the Great Northern Hospital, where he recovered. He was sent to the Convalescent Home at Slough, where he remained in good health. While there, however, a lump in the left iliac fossa was discovered, which had not previously been observed. The child was examined by himself, by Dr. Sutherland, and he believed also by Dr. Guthrie, and the nature of the tumour was a matter of dispute. It was not a fæcal concretion, as the ordinary methods did not remove it. On inquiring carefully into the history the mother said that the child was aware of something flopping up and down in his inside six months before the illness.

He believed the movable portion of the tumour was a floating kidney, which might possibly have been twisted so as to develop the symptoms. He had brought the child for diagnosis as to the nature of the tumour; and secondly, to determine whether the lump could have been related to the symptoms he had described.

(*Exhibited December the* 11*th*, 1903.)

Discussion.

Dr. Edmund Cautley said Dr. Morison had not stated how movable the lump was. He, Dr. Cautley, thought a kidney in that position should be called a floating kidney and, if it were so, he would expect it to move freely about and even across to the other side. But the lump seemed more or less fixed. He thought the child had had an attack of nephritis unconnected with the lump, and that the lump was possibly a tuberculous mass.

Dr. G. A. Sutherland said he had not had time to examine the case that day, but he could well recall it. When he first saw it he thought the lump was a fæcal accumulation. He was interested in the question of movable kidneys in children, and he did not think it was a common condition. But he would support the theory of Dr. Morison that the prolonged attack was possibly due to movable kidney. Several such cases had been sent into the Paddington Green Hospital as acute appendicitis. On investigation one could not say there was not appendicitis owing to the rigidity on the right side of the abdomen, but after a time the symptoms subsided and a movable and tender kidney was discovered. Some of the cases had recurrent attacks. With regard to the albuminuria, he asked if Dr. Morison had noticed whether it was of the postural or cyclic form.

Dr. Septimus Sunderland considered the swellings to be tuberculous. The swelling which was situated highest in the abdomen did not feel of the consistence of kidney, nor was it in the least tender when firmly squeezed.

Dr. Percy Lewis (Folkestone) said he saw a case in which a tumour was discovered after recovery from intestinal obstruction. No diagnosis could be arrived at, but it was agreed that the tumour should be removed. On removal it was found to be a dermoid cyst.

Dr. Leonard Guthrie said he had only examined the case cursorily, but he felt convinced that it was a movable kidney on the left side. He was not clear as to the nature of the swelling below, but thought it might be a tuberculous mass, to which the movable kidney was partly adherent.

Dr. Alexander Morison, in reply, said he did not consider the albuminuria was postural, because the child was in bed the whole time. The improvement in the condition, the increased urination, and the disappearance of the albumin, all occurred before the child got up. He agreed with Dr. Cautley's remarks as to the general mobility of the floating organ. If it was kidney, he thought it had been tied down by inflammatory adhesion, which would limit its excursion. He agreed with Dr. Guthrie that there was probably a lump present in addition to the kidney. The general health seemed good, there had been nothing to indicate acute nephritis. The absence of blood, the rapid disappearance of the large quantity of albumin, and the sudden increase of urine seemed to exclude an active inflammatory condition. Nothing had been found *per rectum*.

17. A CASE OF EXOPHTHALMIC GOITRE IN A CHILD AGED 12 YEARS.

By ARNOLD LAWSON, M.D.

The chief point of interest in this case lies in the extreme youth of the patient. Very few cases of Graves' disease commencing in childhood before the onset of adolescence have been recorded. The patient is a well-nourished girl with a good family history on both sides, and was a healthy child until the onset of the disease. She has had a few of the ordinary ailments of children—such as measles and scarlet fever—but they were not of a markedly severe type, nor were they followed by complications of a serious nature. She presents the typical characteristics of a patient in an advanced stage of Graves' disease, excepting that she has never lost flesh and shows no trace of anæmia. The enlargement of the thyroid, which is very marked, was not noticed until six months after the exophthalmos, which was the first symptom to attract the parents' attention. The urine has so far shown no abnormality, but the child has only been under observation for three weeks. No cause can be assigned for the onset

of the disease. The child has always been of rather a neurotic type, and was easily frightened by being left alone in the dark and by other similar childish fears, but she has never had any severe shock, nor has she ever had fits or shown symptoms of chorea. There is no history of insanity in the family. Though the physical signs are so prominent her general condition is excellent, and the prognosis may be regarded as good. Regarding treatment this should, in my opinion, be conducted on purely medical and hygienic lines, and no operative interference with the thyroid gland, or cervical sympathetic is either expedient or justifiable. (*Exhibited December the 11th*, 1903.)

Discussion.

Dr. James Taylor agreed that it was a very interesting case because the patient was so young. He had only once previously seen the disease in a child at that time of life.

Dr. Robert Hutchison said the association of exophthalmic goitre with disorders of the pelvic organs in women had always been of considerable interest, and he asked whether there was any abnormality in those organs in the present case. Possibly the reason of the rarity of the disease in children was, that the pelvic organs at that age were still in a state of functional inactivity. Had there been abnormal early menstruation in the present case?

Dr. A. E. Sansom asked whether any cause was known for the condition in the child. He had seen the disease in a child, aged 12 years, who had had influenza. The association mentioned by Dr. Hutchison was very interesting, and was undoubted. Still, that might not obtain in children. He did not much like the term exophthalmic goitre; he preferred the old-fashioned name Graves' disease. Tremors were very much in evidence in these cases, and there was much nerve excitability and gastric derangement. The tachycardia nearly always preceded the other symptoms. The rate of the heart in the present case was not very considerable. The condition was often asssociated with von Graefe's sign. He believed there was something zymotic about it as a rule.

The Chairman (**Mr. Sydney Stephenson**) asked whether the prognosis in children with such a condition differed from that in grown-up people.

Dr. G. A. Sutherland asked what treatment Mr. Lawson proposed. Possibly, as a surgeon, he would excise half the

thyroid, but he (Dr. Sutherland) had not been led to believe very much in surgery in such cases. With regard to operations on the sympathetic ganglia in the neck, the fact that the abdominal symptoms were as striking as the thoracic and ocular symptoms was very important as contra-indicating local treatment. In Mr. Lawson's patient there was marked pulsation of the abdominal aorta, and she had suffered from attacks of diarrhœa, such as were characteristic of Graves' disease.

Mr. Arnold Lawson, in reply, said he did not examine the pelvic organs, but the menses had not commenced. No cause for the condition had been elicited. The child did not come of a markedly neurotic stock; the only sign of a neurosis was the occasional waking up in the night screaming. There was no history of fits, epilepsy, or insanity in the family. He had never tried operative treatment for the condition, but he was aware that thyroidectomy had in many cases been followed by death, for there was a mortality of over 12 per cent. in the recorded cases. Perhaps excision of the cervical sympathetic seemed to be more rational than thyroidectomy, but in a case with much pulsation of the abdominal aorta and diarrhœa he did not think excision of the cervical sympathetic would be of any use.

18. A CASE OF MEDIASTINITIS.

By E. P. BAUMANN, M.D.

The case was that of a boy, aged $7\frac{8}{12}$ths years, who, in July, 1903, had an attack of rheumatism complicated by endocarditis and pericarditis. From this he appeared to have recovered, when, in September, his parents noticed puffiness of the face and swelling of his abdomen. On admission into the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Penrose, the condition was as follows: Well-nourished child. No cyanosis or respiratory distress. Puffiness of face; no œdema of legs or scrotum. Veins in neck distended, but no regurgitation. Thorax: some impairment at the base of the right lung; no adventitious signs. Heart somewhat enlarged; cardiac sounds muffled, but no murmurs

heard. The abdomen was very full and contained a large quantity of free fluid; spleen not felt; liver seemed to be enlarged. Urine acid; sp. gr. 1030; no blood, albumin, or tube casts. During his stay in hospital slight œdema of the legs and scrotum appeared on one occasion, but soon subsided again. Paracentesis had to be performed on several occasions for the relief of the abdominal distension. Relief of the tension, which made no difference to the state of the veins of the neck or of the heart, enabled the liver to be definitely found to be considerably enlarged. The organ was very soft, although its surface appeared to be rough and irregular. The urine on no occasion was found to contain albumin. Examination under the X-ray screen threw no further light upon the lesion.

In view of the history and clinical symptoms a diagnosis of adhesive mediastinitis was made.

The boy himself was in good general health.

(*Exhibited December the 11th*, 1903.)

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19. A TUMOUR AT THE ROOT OF THE TONGUE FOR DIAGNOSIS.

(? A RETENTION CYST OR A MISPLACED THYROID.)

By GEORGE CARPENTER, M.D.

L. R—, aged 14 years, has a tumour in the mid-line of the tongue at the back at its base of attachment about the size of the top of the thumb. It has a smooth surface and a somewhat elastic feel, but there is no fluctuation.

The tumour was discovered accidentally by her medical adviser, Dr. Frank Collie of Balham. It has given no cause for anxiety, and she has passed through an attack of scarlet fever without danger.

In association with this tumour is the fact that the middle lobe of the thyroid cannot be felt in the neck, nor

is it possible to satisfy myself that the lateral lobes of the thyroid are present. Apart from this she is a perfectly healthy girl.

Is this tumour a retention cyst or dermoid of the thyro-glossal duct? I should like to ask the members whether they think any importance can be attached to the absence to palpation of the thyroid gland in the neck, and whether if that be the case the tumour at the back of the tongue can be the isthmus of the thyroid gland which has not sunk to its proper level. If that be so, whether the thyroid is solely represented by this tumour at the back of the tongue. In my experience, the fact that the thyroid cannot be felt in the neck is not a sufficient warranty for assuming that it is not in its proper position.

Perhaps an opinion will be forthcoming as to the nature of the tumour, and as to the advisability or not of operative interference. (*Exhibited December the 11th*, 1903.)

Discussion.

Dr. Dan Mackenzie (Leytonstone) said the case was interesting because of the occurrence of the tumour in a similar situation to that of the lingual tonsil, which was subject to enlargement, and when that happened it was liable to be excised. If the tumour should happen to be a thyroid which was in functional activity, its removal might be fraught with danger to the child. Excision of a small portion of the growth and its examination might throw some light on its nature.

Mr. Francis Jaffrey thought the tumour was in connection with the upper end of the thyro-lingual duct. He said there is a specimen in the Museum of the College of Surgeons exactly like it, but not so large.

Dr. Frank Collie (Balham) (introduced) said he had known the child four years, and until one and a half years ago there was no question of anything wrong with the tongue. At the latter time, however, she began to menstruate, and two months later the tumour was noticed and the chin was thrust forward, which might have been due to the effort to get a freer passage of air. He thought the tumour was adenomatous, and Mr. Butlin, who had seen the patient, thought it was a transposed thyroid. Mr. Butlin said he could not make out the isthmus or either of the lateral lobes in their normal position, and he deprecated any attempt at excision. The patient had also seen Sir Felix Semon, who did

not express any definite opinion, but practically concurred with Mr. Butlin, that it was a transposition of the thyroid to the dorsum of the tongue. As the child suffered no inconvenience, he thought that it should be left alone. The tumour had remained in precisely the same condition since it was first noticed and gave very little inconvenience.

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20. AFTER RESULTS OF POLYNEURITIS.

By LEONARD GUTHRIE, M.D.

The patient is a boy, aged 13 years. He was admitted into the Children's Hospital, Paddington Green, on October the 6th, 1896, with the following history. Twelve days previously he had suddenly lost power in his hands and arms; in a few hours his legs became similarly affected. Two days later, he was unable to walk. He complained of headache and pains in his limbs when handled. He had been slightly delirious at night, and the bowels had been relaxed. His previous health had always been good, and there was no history of diphtheria or any form of sore throat. On admission he was healthy looking, and well nourished. The eyes were normal, except for slight nystagmus on looking to the left. The palate was unaffected, and the voice was natural. He was unable to sit up or stand without support, and he could not walk at all. The movements of the upper extremities were all weak, and the hand grasp so much so that he let objects slip through his fingers, and could not feed himself. The legs below the knees were bluish, cold, and tender on pressure. He screamed with pain behind the knees when made to stand. There was foot drop on both sides, and power of dorsiflexion of the feet was much impaired. The extensors of the legs and feet did not react to faradism or galvanism. The knee-jerks and plantar reflexes were absent. Otherwise, he seemed well.

The treatment was by massage and galvanism, together with daily injections of liquor strychninæ, beginning with one minim increased by half a minim every other day until the dose reached 10 minims. By November the 4th, 1896, he was able to sit up in bed and to feed himself, but could not stand alone. The extensor muscles of the legs had wasted, and also the thenar and hypothenar eminences in both hands to a slight extent. Henceforth, he gradually improved, and by the beginning of February, 1897, he could stand and walk alone, and when discharged from hospital, the only remaining symptoms were loss of knee-jerks, and slight commencing contraction of the tendo Achillis on both sides.

He was then lost sight of for six years, but was brought to hospital in February of the present year, with considerable contraction of the tendo Achillis on both sides, and some amount of talipes varus. This condition had rendered walking increasingly difficult. The tendo Achillis was divided on both sides by Mr. Euan Stabb, with considerable improvement in the patient's walking powers.

At present the muscles of the lower extremities are small but fairly firm; none of them is paralysed. He can dorsiflex the feet fairly well. There is a marked condition of pes cavus on both sides. The great toes are hyper-extended at the metatarsal joints, and flexed at the others. There is some degree of talipes varus, and the appearance of the feet strongly resembles that of Friedreich's disease. The knee-jerks and plantar reflexes are absent, but there is no loss of sensation, nor scoliosis nor nystagmus. His gait is awkward and shuffling but not ataxic. The thenar and hypothenar eminences in the hands are slightly wasted, he is clumsy in using his hands, but not ataxic.

Mentally, he is deficient. He goes to a school for backward boys, but learns very little. He can read easy words but cannot write. He is fond of drawing and painting in water colours, but shows no talent in either. His moral sense is dormant. He steals food and any small articles about the house to sell them for a few

pence to boys in the street, and lies like a Trojan when taxed with the thefts. He is also passionate at times, and will swing his younger sisters round by their hair when they provoke him. The "hump" feet, and loss of knee-jerks are suggestive of Friedreich's disease, but he has no other symptoms of that complaint. Spastic paraplegia may be excluded by the absence of the knee-jerk and of spasticity; infantile paralysis by the symmetry of the deformity and absence of definite paralysis of any muscles; myopathy, in all its forms, by the history of onset. Therefore, the condition is probably due to the after results of an attack of polyneuritis from an unknown cause occurring seven years ago.

(*Exhibited December the 11th*, 1903.)

Discussion.

Dr. James Taylor said he saw the case six years ago, at the time of acute illness, and he could not help thinking that Dr. Guthrie, by a process of exclusion, had arrived at the most likely diagnosis. The only suggestion he would add to Dr. Guthrie's remarks was that the child might have suffered from some general toxic condition six years ago, which affected the anterior horns and the peripheral nerves as well, but that the toxin also affected the highest centres and had thus caused the mental condition which was present. As to prognosis, he gathered that the child was not getting worse, and the contracture which was remedied by operation recently, was present in a slight degree when the child left the hospital in the first instance. As to the possible future mental condition, he did not feel inclined to be sanguine.

Dr. Fletcher Beach said he did not question the boy, but he seemed defective in intelligence. One often met with loss of moral control in such cases, though the children were sometimes quite intelligent. He thought the moral nature in the present case had not been properly developed.

Dr. Leonard Guthrie, in reply, said he agreed with Dr. Taylor that the condition was due originally to some form of toxæmia, not of the nature of anterior poliomyelitis, but a toxæmia affecting the anterior horn cells, as well as the peripheral nerves. His own feeling was that the boy would become a derelict in a lunatic asylum or a prison.

21. A CASE OF CIRRHOSIS OF THE LUNG.

By GEORGE CARPENTER, M.D.

Lily C—, aged 6 years, attended at the North-Eastern Hospital for Children in November, 1903. She had been ailing since three years of age, when she had pneumonia, and at that time she was ill for two months. She had whooping-cough at the age of twelve months. She now complains of shortness of breath and cough, which is worse of a morning. She does not expectorate much, nothing greater in quantity than a large-sized marble. The breath is never offensive. Her maternal grandfather died of phthisis, and a maternal uncle also. Her mother is thin and miserable-looking, but she is not considered to have phthisis.

On examination the child has a puffy-looking face, but there is no clubbing of the fingers or toes.

The left lung is dull all over, back and front. The breath sounds are vesicular, and very deficient at the extreme base.

The right lung is hyper-resonant, and the breath-sounds are extra-puerile. The left lung is pushed over somewhat by this; in front the resonance encroaches to the extent of a finger's breadth or more from the level of the fourth costal cartilage upwards. Behind the resonance encroaches on the left side to a finger's breadth beyond the spine.

The heart's apex beat is not displaced; pulsation is also evident in the second, third, and fourth interspaces. The heart sounds are very loud behind.

The other organs are healthy.

There are no tubercle bacilli in the sputum, which is muco-purulent, nummulated, and small in quantity.

(*Exhibited December the 11th*, 1903.)

22. AORTIC STENOSIS IN A GIRL AGED 11 YEARS.

By LEONARD GUTHRIE, M.D.

The patient, a well-grown and well-nourished girl, was admitted into the Children's Hospital, Paddington Green, on November the 23rd, 1903.

Except for pneumonia and pleurisy at the age of three, and subsequently, pertussis and morbilli, she had always been well until five weeks previously. She then complained of headache and pain in the left side and legs. In two days she was well, but a fortnight later the pains returned, and she was brought to hospital with follicular tonsillitis. She is the seventh of nine children, of whom five have had rheumatism, and all have enlarged tonsils. There was also a strong history of rheumatism on the mother's side.

On examination, there was considerable hypertrophy of the left ventricle. The impulse was forcible and heaving. The apex beat was in the sixth space just outside the nipple line. The first sound at the apex was laboured, and a faint systolic bruit could be heard accompanying it, and conducted a short distance into the axilla. A slight thrill was also perceptible preceding and accompanying the heart's impulse. At the base there was a much louder rough systolic bruit. It was heard at the second right intercostal space close to the sternum, but its point of maximum intensity was in a similar position in the left intercostal space. The bruit was conducted into the carotids and the subclavian arteries on both sides, and could also be heard over the back, and loudest midway between the left scapula and spine.

The aortic second sound was ringing and perhaps impure, but there was no definite aortic diastolic bruit, nor evidence of regurgitation. The pulse was small, regular, and quick (112 to 120). The sphygmograph gave

no evidence of high or low tension, but the radial artery seemed small and contracted.

After admission she had another attack of tonsillitis, but without articular symptoms.

Remarks.—The case was one in which difference of opinion might be expected. Congenital malformation of the heart had been suggested as a cause of the systolic basal bruit. But the history, and strong evidence of rheumatism in the family, were in favour of the presence of endocarditis. The fact that the maximum intensity of the basal systolic bruit was in the second left intercostal space might be held to point to stenosis of the pulmonary artery. But in children the systolic bruit of aortic disease was usually heard loudest in the so-called pulmonary area, and in this case there was no evidence of pulmonary obstruction. The left ventricle was hypertrophied, not the right.

For these reasons the diagnosis of aortic stenosis, which might probably lead to incompetence of the valve, had been made. (*Exhibited December the* 11*th*, 1903.)

Discussion.

Dr. Theodore Fisher (Bristol) said he had not seen the case, but congenital aortic disease was rare. A considerable number of cases had been recorded in Dr. George Carpenter's book. In one case he saw, the mother suffered from rheumatism while pregnant, and the cardiac lesion was like aortic stenosis in an adult following rheumatism. The sigmoid valves were joined together and there was a diaphragm with a small aperture in the centre. Congenital disease of the aortic valves was not so rare as might be supposed, neither was the acquired disease in children. He had met with two, if not three, examples in the post-mortem room, in one of which the stenosis was due to a curious morbid anatomical lesion, *i.e.*, instead of the valves being adherent together by inflammation, there was a band just below the centre of the valves running round and forming a diaphragm almost independently of the cusps of the valves themselves. He had seen that not only in children, but in adults.

Dr. Edmund Cautley thought it would be a great advantage if Dr. Guthrie would bring the case again in a year's time. At present he did not disagree with his diagnosis, except that there might be a diastolic murmur as well. He believed it was

not congenital, but due to rheumatic endocarditis of a valve, with roughening of the valves, but perhaps no narrowing of the orifice. He thought there was a diastolic murmur, and if the child could be seen in a year's time one might more accurately ascertain the degree of contraction or regurgitation.

Dr. C. W. Chapman said the case had a short pre-systolic murmur, and a distinct thrill pre-systolic in time could be felt.

Dr. Robert Hutchison said there was no doubt of the presence of a mitral lesion, and it seemed to him there was a pre-systolic and systolic thrill, and that the extremely loud second sound at the base was the accentuated pulmonary second sound of mitral disease. That was what one would expect, whether there was an aortic lesion or not, because if it was rheumatic in origin the probability of the mitral valve escaping altogether was very small. He was not prepared to say whether there was aortic stenosis, but it seemed to him there was no thrill to be felt at the base, although a murmur was heard well in the arteries of the neck. The pulse tracing included in the notes did not suggest aortic stenosis, so Dr. Guthrie would be doing a service by bringing the case again a year hence.

Dr. A. E. Sansom also hoped that the case would be brought again at a future meeting. One heard that there was a suspicion of a diastolic murmur. One heard from Dr. Chapman that there was what might possibly be a Flint murmur due to aortic causes, not to mitral, and then one heard from Dr. Hutchison that there was undoubtedly a mitral affection. So it seemed more likely that rheumatism was the cause, and that it was a complex condition of endocarditis involving many valves or portions of endocardium.

Dr. Alexander Morison said there were features in the case which pointed to the involvement of the pulmonary orifice. He admitted there was a thrill over the apex which was not constant. The hypertrophy of the heart was not great, and he did not see it at the stage at which the apex beat was found in the sixth space. The bruit was distinctly more pronounced on the left of the sternum, which might be the case with aortic bruits, but it had a peculiar venous resonance and hum. The pulse tracing did not suggest to his mind aortic disease, and he was not certain that it was not a case of pulmonary arterial anomaly.

Dr. G. A. Sutherland thought there was a double murmur at the base; and the humming character to which Dr. Morison referred he did not think was produced in the aorta. He suggested it was congenital, due to a patent ductus arteriosus. He asked whether there was any history of blueness in the patient when an infant.

Dr. Leonard Guthrie, in reply, said he would, as suggested, bring the case again at some future time. He remained of the same opinion as before. There was no history of cyanosis.

23. SOME CASES OF LYMPHANGIOMA.

By ALBERT CARLESS, M.S.

It has fallen to my lot to treat a number of these growths during the last few years, and inasmuch as their characters and peculiarities are none too clearly described in text-books, and my cases have ranged over most of the different types assumed by them, it has seemed worth while picking out some of the more characteristic so as to give an idea of the whole subject.

It is a little difficult to draw an exact line of demarcation around growths which should merit this title; between them and the lymphangiectases a somewhat large group exists, of which it is almost impossible to say to which class one should assign the individual members. Theoretically, one looks on a lymphangioma as a tumour built up of *newly formed* lymphatic vessels, whether or not they retain their tubular characters, whilst the lymphangiectases are conditions due to the distension of *pre-existing* lymphatics, and the latter are usually associated with interstitial overgrowth of the tissues involved, constituting in the extremities conditions akin to elephantiasis. The difficulty in particular arises with the unilocular or multilocular cysts, which form the majority of cases known as cystic hygroma; whilst the term lymphangioma is usually applied to these, it is, to my mind, quite an open question whether it is strictly merited.

I propose to lay before you the characters of five cases. Of these, one was purely cutaneous; two involved skin and subcutaneous tissues; three were typically cystic; and the last was a solid growth, but covered by and associated with a marked cutaneous development.

Case 1.—The patient was a child under twelve months of age, of poor physique and badly nourished. It was affected by a patch of lymphangiomatous tissue upon the

temple, the size of a shilling or a little more. It presented the typical appearance of what may well be termed a *lymphatic nævus*. The patch was of a brownish-yellow colour; the surface was slightly irregular and papillated, and on examination with a lens one could see that each of the enlarged papules of the skin was the seat of a dilated lymphatic covered with epithelium. There were a few enlarged blood-vessels in the neighbourhood. As the condition was not increasing in size, and as the child's nutrition was peculiarly defective, I advised that it should be carefully watched and not dealt with at the time; any increase would determine active treatment, in the shape either of destruction by the actual cautery or excision.

In the third and fifth of the cases here recorded, a similar condition was present in association with deeper lymphangiomatous manifestations, but was spread over a much more extensive surface. Not infrequently patients scratch and irritate the affected areas, giving rise to a flow of lymph from the surface, and perhaps followed by attacks of inflammation which may lead either to extension or to cure.

This lesion is sometimes associated with a condition in which the lymphatics are distended to such a degree as to constitute vesicles as large as a half pea, but the latter may also occur without the former. These *lymphatic varices* are by no means uncommon, and are best treated by complete extirpation if they be few in number, or by opening them and cauterising the exposed surface. A very good illustration of this "Lymphangioma circumscriptum" appeared in the 'British Medical Journal' on June the 3rd, 1893.

Lymphangiomata of the subcutaneous tissues similarly present themselves under two chief manifestations viz., the tubular, and the cystic or cavernous. In the former, as in the hæmangiomata, the newly formed vessels remain as capillaries, and constitute soft pulpy or spongy swellings, which can sometimes be reduced in size on pressure. In the cavernous type, cysts of greater or smaller size are

found, associated or not with the spongy tissue just mentioned, and filled with lymph, to which a greater or less quantity of recent or old blood is not infrequently added. On the whole, one is inclined to think that the purely cavernous without admixture of spongy tissue is more frequently seen. As an illustration of this, the following case may be noted.

Case 2.—D. M. S—, aged 5 years, was admitted into King's College Hospital on September the 9th last for a cystic swelling situated in the lower part of the right cheek and upper part of the neck. It had only been noticed three months previously, but during the month preceding her admission it had increased in size very considerably. It extended upwards as high as the external auditory meatus, downwards to the level of the thyroid cartilage, backwards to the anterior border of the sterno-mastoid, and forwards nearly as far as the middle line. The swelling was tense and elastic, apparently loculated, and the skin over it was normal and moved freely; the growth appeared to be more or less attached to deeper structures. The limits of the swelling were quite sharply defined, and it was obviously of a cystic nature. It was operated upon on September the 11th, and on incision proved to be a thin-walled multiloculated cyst, containing serum mixed with blood, which from its colour had been extravasated into it some little time. The various loculi penetrated deeply amongst the structures of the neck, and were in close proximity to the vessels; the lingual and facial arteries projected into the cavity, and were only separated from it by the endothelial lining. One loculus ran up under cover of the mandible into the pterygoid region. It was impossible to dissect out in its entirety the whole of the lining wall, and hence those portions which could not be thus dissected away were well scraped by a sharp spoon. The wound was closed by a continuous suture and healed by first intention.

I look on this growth as a pure cystic lymphangioma

though why it appeared in this situation, and why it developed so suddenly and rapidly, it is impossible to say. Its definite limitation rendered it very suitable for treatment by operative measures. Not a few growths ordinarily termed cystic hygroma are of this type; they are perhaps more frequently composed of a congeries of smaller cysts than of a single one, but they are frequently quite amenable to operative treatment. It is by no means unusual to see a hæmangiomatous element also present, and this association may explain why the fluid contained in them is stained or mixed with blood. In one instance which I saw years back under the care of Lord Lister, it was possible to reduce the size of the mass by pressure to a considerable extent, and as soon as the compressing force was removed the tumour swelled up to its former dimensions.

Quite distinct in their features from this, although still cavernous lymphangiomata, are the two next cases.

Case 3.—H. G—, a boy aged 6 years, came under my care at St. John's Hospital, Twickenham, about two years ago. He was the subject of a large cystic swelling on the left side of the posterior thoracic, and abdominal walls. This had existed from birth, but had increased in size rapidly during the previous few months. The mass extended from the mammary line in front nearly to the middle line behind; from above downwards it was about 4 inches in diameter, and projected outwards to such an extent that it resembled in size a large cocoa-nut. Several cysts could be detected, but the main central one was of considerable dimensions. The skin covering the mass was adherent to it, and in part the seat of a cutaneous lymphangioma resembling that described in Case 1. The growth appeared to be more or less fixed to the thoracic parietes. I dealt with it in two operations. In the first the incision was a straight one placed across the centre of the mass, and extending from an inch and a half external to the left nipple obliquely downwards and backwards nearly to the

middle line behind just above the top of the sacrum. The main cyst was thereby opened and found to contain serous fluid. Many other cysts were present, but in addition to this was a considerable quantity of a soft spongy tissue which oozed lymph when cut into, and was much of the same character as the soft spongy hæmangiomatous tissue found in ordinary nævi. This, together with the lining wall of the main cyst and the greater portion of the affected skin, was dissected away; but it was found that the soft nævoid tissue extended into and had infiltrated the superficial muscles covering the thorax and abdomen, and it was necessary to encroach on these to a considerable extent in order to get clear of the growth. There was a good deal of bleeding in this procedure, and therefore it was thought wise not to attempt the total extirpation of the outlying portions of the mass towards the middle line. The patient recovered from this operation perfectly well, and some time later a second operation was undertaken in order to remove the remains of the tumour, which had once again started growing. This time one made a curved incision extending from the upper end of the scar to nearly the lower, and reaching back as far as the middle line. This large flap was dissected up, and all the remaining portions of the growth were removed. The wound healed by first intention. I saw the patient a few weeks back, *i. e.* eighteen months after the second operation, and found that the condition was perfectly satisfactory; there was no sign of any recurrence, and the skin only in one spot showed evidence of the preceding lymphangiomatous condition which had involved it.

Case 4 was a less satisfactory one of a similar type, in which, however, a fatal issue ensued in spite of extensive operative interference.

Mary E—, aged 3 days, was admitted into King's College Hospital under Mr. Rose on March the 3rd, 1902, with a large cystic swelling beneath the left side of the jaw, extending from the symphysis menti to the mastoid pro-

cess. The skin was not adherent over the mass, and several distinct cysts could be made out. The growth increased rapidly in size, several of the cysts appearing to merge into one another under observation, and the mass soon began to project further backwards behind the sterno-mastoid. On March the 10th Mr. Rose operated, making an incision more or less parallel with the ramus of the jaw over the more prominent cysts. The skin was separated from them, and the cysts opened. They contained yellow serous fluid and a substance of jelly-like structure, probably due to coagulation of the lymph *in situ*. The greater part of the mass was removed, and its pedicle ligatured. The wound was closed and the child did well, but the notes state that on March the 20th, before the child was discharged, the swelling under the chin was again commencing to enlarge. The fluid contained in the cysts is reported by Dr. Grünbaum to contain 0·4 per cent. of albumin. The wall of the cysts was composed of connective tissue, in parts undergoing myxomatous degeneration; the cyst cavity was not lined by epithelium. The solid material in the cysts was said to be organising fibrin.

The child was admitted a second time under my care a few months later. The growth had then attained considerable dimensions, extending under the chin as a projecting rounded mass, and also occupying both triangles of the neck. The child's nutrition had by this time begun to suffer, and although operation was essential, one was a little afraid as to what the result would be. A large incision was made, extending the whole length of the lateral aspect of the neck, and it was early seen that the sterno-mastoid was extensively invaded by the growth, and that a removal of its whole thickness in a portion of its extent was required—in fact, there were very few muscle-fibres left towards the centre. The growth had also attacked the parotid gland, and its removal from this region necessitated the division of a portion of the facial nerve. In spite of the very extensive dissection, it was impossible to remove the whole mass, especially under the chin. When

the operation was finished, the whole of the deeper structures of the neck were laid bare, and one had to place the skin-flap down on the internal jugular vein with no other structure intervening. In spite of all things, the wound healed satisfactorily, and the child was sent out of hospital; but, unfortunately, the side of the face remained partially paralysed.

The ultimate result was that the tumour grew once again, burst through the skin and became infected. The child was admitted to hospital for a third time on January the 10th this year, and died of septic absorption.

The most important feature in each of these two cases is the tendency obviously existent in them to infiltrate surrounding structures. The sterno-mastoid in one case, and the superficial abdominal muscles in the other had been invaded, and their tissues infiltrated and destroyed; the parotid gland in Case 4 had also been attacked. In the former case the parts involved were of less importance, and therefore could be freely dissected away; but when the deeper tissues of the neck are affected, serious results may follow, and life itself may be destroyed. This tendency to invade and infiltrate has been noticed by other observers, and must ever be kept in mind in advising as to treatment. The tissue is plainly of a lowly organisation, and it is quite an open question whether there is not some added sarcomatous element present. A similar instability of the hæmangiomata has also been noticed.

Mr. Betham Robinson related a case somewhat similar in which the parotid region was invaded by a growth of this nature, though there it was simply cystic and not of the spongy type. An operation was undertaken, but only a portion could be removed. The growth recurred, and the child subsequently died of asthenia ('Path. Soc. Trans,' 1896). Mr. Spencer has also reported to the Medical Society a case of diffuse lymphangioma of the lower extremity, and has referred to some others of a similar type; but they do not seem quite to fall into line with cases such as I have referred to, and are more like diffuse

lymphangiectases ('Med. Soc. Trans.,' 1892, p. 133). Zuccaro ('Puglia Medica,' 1894, Nos. 8 and 9) also relates a case of diffuse cystic disease of this type in the upper arm and thorax of a child seventeen days old, where the connective tissue was riddled with cysts, and gives a careful account of the histology. Complete removal was impossible, and death occurred as the result of septic processes starting in the wound, which could not be completely closed. Vautrin ('Revue de Chirurgie,' 1898, p. 1128) relates a case of mesenteric lymphangioma which he discovered post mortem. The growth had invaded the muscular tunics of the stomach, and was surrounded by agglutinated coils of intestine. He emphasises the gravity of this invasion, and looks on it as analogous to that of a malignant tumour.

Operations for partial removal must therefore always be avoided, if possible, but there are not a few cases on record in which a good result was subsequently obtained. D'Arcy Power ('Brit. Med. Journ.,' December the 4th, 1897) relates one in which spontaneous disappearance of the mass occurred subsequently, owing to an attack of inflammation in the part left behind; but, as one has indicated by the cases referred to above, this inflammation may not always lead to a satisfactory issue.

The last case I have to describe was an interesting one in which there was a solid fatty growth on the anterior thoracic wall, adherent to the skin which covered it in, and this in turn was invaded for a good portion of its extent by a cutaneous lymphangiomatous development, similar in characters to that which I alluded to in my first case.

The child, M. S—, was 11 years of age, and the growth had been noticed since she was two years old. It increased gradually in size, but gave no inconvenience till about two and a half years previously, when she was operated on by Mr. Stanley Boyd at Charing Cross Hospital. The mother stated that there was no sign of the mass after this procedure, but that it commenced to grow again shortly

afterwards. On her admission into King's College Hospital in August, 1901, the swelling extended vertically from the second to the fifth rib inclusive, and from half an inch to the right of the middle line to well into the left axilla. It projected forwards two or three inches, constituting a tumour rather larger than a fist; and towards its lower part could be seen the rudimentary breast, which was quite independent of it. In consistence it was soft, slightly lobulated and irregular; freely movable on the deeper parts, but the skin was adherent over it. Enlarged veins coursed over the mass, and the scar of the former operation ran transversely across its lower border. The skin over the greater part of it, and especially towards the axilla, was slightly warty and papillated, resembling in colour and appearance the lymphangiomatous developments already described. No enlarged glands were to be felt in the axilla, and the growth was quite painless.

The whole mass was subsequently excised, and as it encroached somewhat closely upon the pectoral muscle, which looked decidedly infiltrated, it was thought better to remove the sternal portion of that muscle, and to carry the incision well into the axilla, from which a somewhat enlarged gland was removed.

The exact nature of this case is certainly open to discussion. The microscopic report as to the cutaneous condition merely stated that it was a papillomatous nævus; but looking to the appearance of the lesion before removal, the total absence of any evidence of circulating blood, and the entire accordance of its characters with those of demonstrated lymphatic nævi, one is forced to the conclusion that it must be placed in that group.

As to the main mass, the report stated that it was merely fat, and that no lymphatic or hæmangiomatous developments were present; it was, however, a large mass, and it is possible that the portion that might have demonstrated its dependence on a lymphangiomatous origin was not examined. That fibro-fatty growths are associated with lymphangiomatous developments cannot be doubted. Mr.

Marmaduke Sheild showed a child to the Medical Society in 1893 ('Med. Soc. Trans.,' vol. xvi, p. 350) who was the subject of what he supposed to be a congenital fibro-fatty growth. In the next year's 'Transactions' (p. 334) the sequel of this case is related. An acute attack of inflammation supervened in the growth, which became enormously enlarged, hot, red, and œdematous, and in spite of careful nursing the child died. Post mortem it was found that the greater part of the growth consisted of solid fibro-fatty tissue, but that towards the axilla it merged into a sponge-like growth, and finally into distinct cysts, the size of peas or grapes, filled with yellow serous fluid. Spontaneous inflammation had occurred, and the child had been unable to withstand the inflammatory fever and exhaustion. I look on my case as akin to this, but that the transformation into a cavernous type of lymphangioma had not been reached. In such developments one sees an analogy to the lipomatous conditions which are sometimes found associated with the hæmangiomata.

(*Read December the 11th*, 1903.)

Discussion.

Mr. Douglas Drew said his experience was limited to cystic angioma and cystic hygroma. One case for which he operated in the neck had features very similar to those Mr. Carless had named, the growth having infiltrated into the sterno-mastoid. He got the tumour away completely. In the second case he was not so fortunate. It was a cystic hygroma, and it extended from the elbow to the axilla, and involved the anterior portion of the chest, including the whole of the mammary region. The child was 18 months old. He operated on the thoracic portion first, removing the whole of the subcutaneous tissue, which was extensively cystic. Then the child showed signs of shock, and he had to stop operating. He attacked the axillary portion a fortnight later, and found the cysts very numerous, and the axillary nerves and vessels ran through the centre of the mass. He had to cease again owing to the child's condition. A fortnight later the remaining portion of the tumour situated on the arm swelled up enormously, became very painful, the temperature was high, and remained so for some fourteen days. Incisions were made into the swelling, but no pus was found, and there

had not been any suppuration previously. The inflammation gradually subsided. He had recently seen the child again, and the tumour remained practically the same. He now had a baby under his care with the same condition, but as the tumour was not growing he did not feel inclined to operate. Their growth he considered to constitute the indication for removal. Mr. Carless's first case seemed to resemble lymphangiectasis of the tongue, and he would like to hear whether that gentleman considered it similar to macroglossia.

Mr. Thomson Walker thought it was doubtful whether all the cases referred to were lymphangioma pure and simple. When one heard of myxomatous and other tissues being present, one thought they must differ from the simple congenital lymphangioma. He did not know of any myomatous degeneration, as mentioned in the paper. Wherever there was myxomatous tissue he thought there was danger of recurrence.

Mr. Albert Carless, in reply, said he did not think there could be any doubt as to the case being a lymphangioma: it was typical, and quite similar in structure to the cases constantly seen of cystic hygroma. It was quite unnecessary to suppose that any other element was present; granting an instability of tissue similar to what one sees in many tumours, the later manifestations could quite easily be explained.

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24. A CASE OF PRESTERNAL DERMOID CYST.

By J. W. THOMSON WALKER, C.M.

Ivy H—, aged 5 years, was sent to me at the North-West London Hospital by Dr. Sutherland.

Since infancy a small round swelling had been present over the sternum. This at first was the size of a pea, but increased steadily until two months ago, when it ceased growing. No similar swellings or other deformities were known to exist in the family.

On examination a smooth round swelling was found about the size of a large horse-chestnut and situated over the junction of the manubrium with the gladiolus sterni. Fluctuation was readily made out and the cyst was not attached to the skin. There was no impulse on cough-

ing and it was thought probable that there was no communication with the interior of the thorax. The cyst was easily dissected out and was found to have no connection with the interior of the thorax. The contents consisted of white grumous material like curdled milk. The cyst wall, when turned inside out, was seen to be lined with epithelium, which showed numerous ridges. No hairs were visible to the naked eye.

On microscopic examination the wall consisted of an outer layer of loose connective tissue and a lining of squamous epithelium raised into ridges. There were no papillæ. Hair-follicles and sebaceous glands were present in fair numbers and there were well-formed sweat-glands.

Dermoids in this situation are usually classed under dermoids of the thorax along with those met with in the mediastinum. I have ventured to exhibit the specimen under the name of presternal dermoid, as the position of these cysts is so exactly similar in each case and there is less chance of confusion with the graver form of intra-thoracic dermoid. These presternal dermoids are not common, but the cases reported in the literature all occupy exactly the same position over the junction of the manubrium and gladiolus. The risk of epiblastic inclusion in the union of the lateral folds of the embryo is apparently greater at this point than at any other part of the mid-line of the body anteriorly.

The only illustrations of these lesions I have found in the literature are in the articles by Bramann and by Cahan. Others have been reported by Chitten and by Clutton. (*Exhibited December the* 11*th*, 1903.)

25. SPECIMEN OF TUBERCULOUS CARIES OF THE UPPER DORSAL SPINE, WITH AN ABSCESS PRESSING ON THE BIFURCATION OF THE TRACHEA.

By DOUGLAS DREW, B.S.

This specimen illustrates one of the rare complications of caries of the upper dorsal region. The child, aged 5 years, had been under my care for some months and was being treated in a double Thomas's splint with a head-piece. According to the mother's statement, difficulty in breathing had been present three weeks, and at times had been urgent, but had improved a few days before admission into the hospital. When I saw the child there was well-marked recession of the ribs, but the dyspnœa was not urgent and there was no lividity. The diagnosis as to the cause of the dyspnœa presented some difficulty, but in the absence of lung disease or evidence of enlarged glands in the neck, it appeared to be more than probable that enlarged mediastinal glands could be excluded and that the symptoms were due to an abscess in connection with the diseased spine.

There were no physical signs in the lungs beyond that the respiratory murmur was somewhat blurred on both sides.

As there did not appear to be any immediate urgency, in that the symptoms had lasted three months and were not pressing, I decided not to operate at once. This proved to be disastrous, as the dyspnœa became urgent early in the following morning and the child died asphyxiated.

The specimen shows the upper dorsal vertebræ, which are extensively diseased and present an angular curvature, the greatest prominence of which is opposite the fourth and fifth intercostal spaces.

Adherent to the front of the bodies of the vertebræ is a

large, thick-walled abscess, which is pressing on the trachea and bronchi. It will be observed that the abscess overlaps the bodies of the vertebrae on both sides almost as far as the heads of the ribs.

If I had at once carried out my intention of exploring for an abscess, by resecting the proximal end of one or more ribs with the transverse processes, no great difficulty would have been experienced in finding and evacuating the abscess; and from the specimen it will be seen that the abscess could have been reached with equal facility from either side of the vertebrae, though doubtless from choice one would have selected the left side.

Although, in this case, the condition could have been successfully dealt with, yet under similar circumstances but with a small abscess insuperable difficulties might be encountered in attempting to reach it.

The chief difficulty in such cases, however, appears to be in determining the cause of the pressure—*i.e.*, whether it is due to a spinal abscess or to enlarged mediastinal glands; and if it is not possible to arrive at a diagnosis with some degree of certainty one would naturally hesitate before undertaking so severe a procedure purely as an exploratory measure.

(*Exhibited December the 11th,* 1903.)

Discussion.

Dr. Theodore Fisher (Bristol) said that although tuberculous glands were common it was very rare for them to be large enough to cause obstruction. He had only met with one case where obstruction of bronchial tubes was caused by glands.

Mr. Douglas Drew, in reply, agreed with Dr. Fisher's remarks. In the one case he had seen he performed tracheotomy, the patient being a 6 weeks old baby. He could not relieve the child without passing a catheter down the trachea.

26. A CASE OF TRANSPOSITION OF THE VISCERA.

By ERIC PRITCHARD, M.D.

The case was one of transposition of the viscera in a girl aged 12 years. The heart was situated on the right side, and the apex beat was about 1 inch outside the right nipple line. The liver and spleen were both transposed. The stomach resonance apparently extended to the right hypochondriac region, which was suggestive that the fundus of the stomach was in this situation and that the stomach was also transposed. The child had been delicate from birth onwards, and when a year old had been admitted to the Westminster Hospital under the late Dr. O. Sturgess. The case had apparently always been regarded as an anomalous one, and the liver had been mistaken for an enlarged spleen. In December last the child was admitted an out-patient at the Victoria Hospital for Children, under Dr. H. D. Rolleston, and the transposition of viscera was discovered.

(*Exhibited January the 15th*, 1904.)

27. A CASE OF CONGENITAL ABSENCE OF THE FOREARM.

By ERIC PRITCHARD, M.D.

The patient, a girl aged 15 years, under the care of Dr. H. D. Rolleston, at the Victoria Hospital for Children, had congenital absence of the forearm. The stump ended about 2½ inches below the elbow in a small excrescence which presented the appearance of a rudimentary thumb provided with a nail. The exhibitor did not regard the case as an ordinary example of intra-uterine amputation, but as one of arrested growth during

an early stage of development by a fibrous band which partially strangulated the growing extremity.

(*Exhibited January the 15th*, 1904.)

Discussion.

Mr. A. H. Tubby, remarking on the case of absence of the forearm, said it was interesting to note that in the stump there were the remains of the upper part of the ulna, and upon it the triceps acted freely. One could see the olecranon moving freely backwards and forwards with the triceps. Also it showed how much more cleanly Nature performed her amputations and how much better the result was than that achieved by surgeons. The stump in the case was ideal, and would gladden any surgeon's heart.

Dr. G. E. Shuttleworth asked if Dr. Pritchard knew what was the cause of the mental backwardness in the case of transposition of the viscera. He understood the child attended a special school. Evidently she was infantile, having the capacity usually met with in a child at 3 or 4 years. Occasionally some indefinite swelling under the skin had been noticed by the mother, and he would be glad to hear whether there was any myxœdematous element in the case which might throw light upon the mental condition.

Dr. C. O. Hawthorne suggested that a skiagram might be taken of the transposition of the viscera case, not to confirm the diagnosis, for the essential facts of the case seemed quite clear, but the skiagram would show whether the arch of the aorta turned to the right or to the left, and this was a detail of some interest. The case of congenital absence of the forearm was of great interest. The nail-like process had many parallels; he had seen at least two cases in which there were processes that might be claimed to be digits. In one of them there were five, and it was easy to appreciate the wider groove which represented the space between the thumb and first finger. Absence of the forearm in such cases was fairly common, and that might perhaps be used as an argument in favour of the view that the condition was due to an actual intra-uterine amputation. The fact that the "accident" occurred at about the same point seemed to show that it did not depend upon developmental impulse so much as upon some mechanical condition. Whether this was an extra-fœtal band, or some trophic change in the skin, was a disputed point. In a case he exhibited eighteen months ago, he tried to show that the constriction was caused by the umbilical cord.

Dr. George Carpenter thought there could be no doubt about the diagnosis in the case of transposition of the viscera. He would like to point out also another congenital deformity in the case of absence of the forearm, viz. a partial webbing of the

index and ring fingers on both hands. He thought that this was strongly in favour of the lacking forearm being due to errors of development and not to supposed mechanical causes.

Mr. Keogh Murphy said he had thought much about cases of congenital absence of extremities, and he regarded the idea of intra-uterine amputation as not quite the correct one. In the present case he would instance the entire absence of cicatricial contraction or retraction of muscles. He believed there were two separate varieties. In one, where the so-called amputation had taken place, the extremity was rounded and the tissues were freely movable over it. In the other class there was some attempt at the representation of the digits at the amputation point. He had been directly interested in the birth of five cases where there was absence of one or other upper extremity, and careful examination failed to reveal any bony remains. It was reasonable to assume that such remains might be absorbed, but one would think that in some cases some remains would be found. Friends of his had investigated in the same way, but had not found remains. He had a case now under observation, which he hoped to bring before the Society later, in which there had been intra-uterine amputation of both feet. In one, Nature had performed the amputation with results more beautiful than one could generally see. In the other there were the remains of the metatarsal bones. In the hands of the same patient there was webbing between the index and middle fingers. There was no sign of bone in these fingers, and the joints were so loose that on flexion the fingers were drawn down into the palm.

Dr. Eric Pritchard, in reply, said he had not a very complete history of the transposition of the viscera child, but some notes he saw, which had been taken some time ago, showed that the child was evidently mentally deficient, and the mother was aware of the fact. Altogether, the child was of a degenerate type. He would have a skiagram taken to show if possible the direction taken by the arch of the aorta. In regard to the case of congenital absence of the forearm, he would have a skiagram taken to show whether there were any rudimentary bones.

28. CASE OF SARCOMA OF THE FIBULA WITH SECONDARY DEPOSITS.

By A. H. TUBBY, M.S.

George L—, aged 2⅞ths years, was admitted into the Evelina Hospital on December 30th, 1903. There are no

points of importance in the family history. The account of the onset of the present affection is as follows: The child had always been healthy until seven weeks ago, and the mother says that then he was hit with a toy-cart on the left leg and she noticed shortly afterwards a swelling. He was seen by a medical man, who ordered poulticing. Three weeks ago he was again hit on the same leg. He also fell and hit his head, and from that time the swelling on the head was noticed.

Present condition.—On the outer side of the left leg there is a firm smooth swelling which is rather diffuse and fades gradually into the surrounding tissues It is neither red nor painful and does not fluctuate, and there are no enlarged veins over it. It is situated 1 inch below the joint and is apparently connected with the fibula, and is oval in shape, and about 3½ inches long by 2 inches wide, the long axis of the swelling corresponding with that of the bone. An exploring needle drew off blood only. On the same side the horizontal group of inguinal glands are affected, and the iliac glands also. They are enlarged and adherent, but not tender. On the right parietal bone on its posterior part there is felt a hard non-fluctuating tumour, and it is seen to be circular in shape and measures 1 inch across. It is apparently incorporated with the bone, its centre is not depressed, and it is firm and resistant. Before admission the child had some vomiting but no rigors. On examination no signs of secondary growth are detected in the lungs.

On January 2nd the patient was very restless and cried a good deal and vomited four times in twenty-four hours. On January 6th the patient did not look so well and both the primary growth and inguinal glands had increased in size. The vomiting continued at times and was quite independent of food. On January 10th it had been noted that the patient had been much quieter and somewhat drowsy during the past few days, but the growth on the leg had diminished half an inch in circumference. One of the most movable of the glands had been dissected on

January 7th, and the specimen showed sarcomatous cells of a spindle shape diffused throughout the gland. Mr. Sydney Stephenson kindly examined the eyes, and the discs were found to be slightly indistinct and the vessels congested. But there were no definite signs of neuritis. The child is now losing his colour and is becoming more apathetic. On the night of January 10th the temperature went up suddenly to 100° F.

Remarks.—The points of interest in this case are: Firstly, the direct connection of the growth with an injury. In the year 1889 the writer, in a paper in the 'Lancet,' pointed out the direct relation of traumatism to sarcoma of bone, a fact not then generally grasped but now widely accepted. Secondly, the growth on the leg and the growth on the head both followed a direct injury to the part and appeared almost simultaneously, and therefore may be looked upon as separate local outbreaks of a specific disorder. Thirdly, the occurrence of fever in cases of rapidly growing sarcoma, while it complicates the diagnosis, is not unknown. The writer had a case of sarcoma of the lower end of the femur in a girl, aged 13, and the temperature reached 102° F., thus inducing one of his colleagues to believe that it was a case of acute periostitis. This was disproved by an incision. Fourthly, no treatment is available in this case on account of the implication of the glands. (*Exhibited January the 15th*, 1904.)

Discussion.

The Chairman (**Dr. Porter Parkinson**) said an interesting feature was, that the circumference of the limb had got rather less, which he supposed simply meant a lessened congestion of the part, but he would like to hear whether the decrease was permanent.

Mr. W. Milner Burgess (Harlesden) asked whether Mr. Tubby had tried Coley's fluid in such cases. He had one case, an adult, in which he thought some benefit followed its use, but the patient would not continue it.

Mr. Thomson Walker asked whether the statement as to the two growths being separately primary was merely a suggestion, or could it be upheld. It was very unusual to find two growths

of a similar character arise in separate parts of the body simultaneously; and it was a difficult matter to prove, because the primary growth in the bone might be quite small, at a time when the secondary deposits were moderately large.

Mr. Lockhart Mummery thought the case suggested that the primary growth might have been in the glands, and that the growths in the head and leg were both secondary. A growth might occur in the glands of the pelvis without giving external signs.

Mr. **A. H. Tubby**, in reply, said the decrease in size was explicable in more ways than one. There was the possibility of lessening of congestion bringing about such a change. But it was also possible to suppose that the growth spread in a horizontal plane and decreased in depth. At one time he had a good deal of experience in the injection of Coley's fluid. Three years ago he injected four cases with it, and the patients would not put up with it. In the first place, they could not be convinced that it was doing good, neither was he convinced, and he had never seen a case in which it could be truthfully said that Coley's fluid had been of any value. Regarding the question as to which of the growths was secondary or primary, or whether one was dependent on the other, he particularly showed the case for the reason of inviting discussion; he thought it might throw some light on what he called the tendency of the child to a general outbreak of malignant disease. It was possible that there were two separate primary growths, though that view was certainly open to criticism. He thought the growth in the leg was primary and that in the head too. Recently a very interesting lecture had been delivered by Mr. Henry Morris at the College of Surgeons on the subject of cancer, and he, Mr. Tubby, could not help regretting that the tendency should be to retrograde and talk of mesoblastic remains. He thought the line for research was in a totally different direction.

29. A CASE OF CONGENITAL UNILATERAL DISLOCATION OF THE FIFTH CERVICAL VERTEBRA.

By A. H. TUBBY, M.S.

Margaret D—, aged 3 months, was admitted into the Evelina Hospital on December 17th, 1903. The patient is the second child; the first child was quite healthy, and

there had been no miscarriages, nor is there any history of syphilis in either parents. The presentation at birth was vertical, the labour was easy, and no instruments were used. The deformity of the neck was noticed the morning after birth on washing the child. No symptoms have been noticed until the last week, when vomiting occurred three or four times a day.

Condition on admission.—The child is somewhat puny and ill-nourished. It cries and is very fretful. At the base of the neck there is seen a shary bony prominence which corresponds to the fifth cervical vertebra On examination under an anæsthetic the summit of the prominence is formed by the left articular process of the fifth cervical vertebra, and the spinous process can be felt to the right of it, but not so prominent. On putting a finger into the pharynx a depression can be felt on the left side of the affected vertebra. The child is also the subject of congenital scoliosis, the curve being to the right in the dorsal region and to the left in the lumbar. A slight rash was noticed on the buttocks on admission, but this readily disappeared without the administration of mercury, and no signs of syphilis are to be seen. The child was placed in bed recumbent with the head of the bed raised, and weight extension of two pounds applied to the child's head. From this time her cries ceased, and she took her food well. An X-ray examination was attempted but nothing could be defined. No efforts of reduction have been made. Finally, there are no signs of paralysis at all, and the bladder and rectum act well. The costal cartilages of the fifth to the eighth ribs are absent on the right side, with sinking in of the chest-wall.

Remarks.—As to the diagnosis. It appeared to lie between congenital syphilitic caries of the spine and dislocation, but against the former there were no signs of syphilis in the parents' history, and, although the child had a transient rash on the buttocks, yet this yielded to care and cleanliness. Then the prominence is limited to one vertebra alone, and this vertebra is twisted so

as to bring its left half backwards and its right half forwards. It is well known that unilateral dislocations do occur, especially in the cervical vertebræ, and that they are not necessarily accompanied by cord symptoms. But it is difficult in so young a child to obtain evidence of root symptoms. As to the origin of the dislocation. The birth was a very easy one and no traction was used, and the question arises, Is the dislocation the result of the congenital scoliosis, that is to say, has one vertebra been partially forced out of place in an unstable cervical spine, unstable because of a compensatory curve in the cervical region? The presence of congenital scoliosis is an exceptionally interesting fact, and the writer has not seen more than five cases in the course of a somewhat extended experience. It is no doubt due to close packing *in utero*, or to the head being unusually inclined towards the left iliac fossa. In nearly all the cases the large curve has been in the dorsal region and to the right. As to treatment. An attempt will be made to reduce the vertebra by traction and manipulation. But it is not likely to be successful, as three months have elapsed since the occurrence of the accident.

(*Exhibited January the 15th*, 1904.)

Discussion.

The Chairman (**Dr. Porter Parkinson**) said he did not know whether, in saying there were no cord symptoms, Mr. Tubby included the condition of the reflexes. He asked whether they were normal. *A priori*, it seemed impossible that there could be such extreme displacement of one vertebra from another without some pressure on the cord resulting, and if so, there would be some sign of it.

Mr. Howell Evans said the case presented itself to him as follows: that the marked prominence felt to the left of the middle line was the unopposed superior articular bearing portion of the lamina of the fifth vertebra, and that the small projection which could be felt almost in the middle line corresponded to the spinous process of the same vertebra. One, therefore, had to account for the depression above the most marked projection. Therefore he regarded it as an absence or displacement of the vertebra above the projection on the left side. Scoliosis was an

accompaniment of it. He knew that scoliosis *in utero* had been discussed, but he did not think any case had yet been reported without partial absence of a vertebra being present. This would explain why, in the present case, there was only half the fourth cervical vertebra, and how consequently the articular surface of the left half of the lamina of the fifth vertebra was trying to get up to the third transverse process, but it could not. There was also, in this child, an absence or malformation of the ribs on the opposite side. So far as he was acquainted with the researches of Ballantyne, he explained the cases by some abnormality in the development of the spinal nerves which passed to those parts. He had seen one case somewhat allied in an adult. Two adults were brawling. The one, getting his head under the arm of his antagonist, was thrown forcibly on to his back; a wrench was given, and a similar condition was produced, namely, unilateral dislocation of the fourth cervical vertebra. He was asked by the medical officer at the infirmary to see the case, and say whether it ought to be sent to a hospital or be treated by himself. As it had a complicated medico-legal bearing, he advised that it should be left alone; it rectified itself with but little mechanical assistance, namely, the head was turned towards the opposite side, and the shoulder pushed backwards, and probably when the spasm of the strong neck muscles relaxed during sleep, there was a return to the normal. He did not think that would result if the same treatment were applied to the present child. If there had been pressure symptoms, it would have shown that the deformity was produced in the act of birth.

Mr. A. H. Tubby, in reply, said he believed the reflexes were normal. There was much to be said in favour of Mr. Howell Evans' view; and he was aware of the possibility of there being absence of portions of vertebræ. He had carefully considered that view. He omitted to quote in his remarks, with regard to the movements of the head and neck, that there was distinct rigidity of the neck laterally, but the movements of the head backwards and forwards were fairly complete. He did not see how it was possible that the absence of the fourth cervical vertebra could make one particular vertebra so prominent, and so cause loss of passive movement. He saw nothing incongruous in the absence of cord symptoms in unilateral dislocation of cervical vertebræ. He would look more for root symptoms than cord symptoms, but the difficulty of finding such symptoms in a child would be appreciated. The constant crying was relieved by extension of the head. He hoped to have a good skiagram of the case taken; and, if successful, he would show it at the next meeting.

Postscript, October, 1904.—A skiagram of the case was taken and proved Mr. Howell Evans' opinion to be correct; the left half of the fourth cervical vertebra was congenitally deficient.

30. A SPECIMEN OF CONGENITAL MALFORMATION OF THE HEART.

By ERIC PRITCHARD, M.D.

The heart was obtained from the case which was exhibited at the November meeting of the Society.* Shortly after this meeting the infant was admitted under Dr. Walter Carr at the Victoria Hospital for Children, suffering from broncho-pneumonia. It died in December, and at the necropsy the following conditions were found: extensive broncho-pneumonia, with slight collapse, and emphysema of small portions of the lung. Regarding the heart, the right and left sides were of similar size and thickness of wall; there was a considerable communication between the ventricles at the unguarded space and between the auricles at the foramen ovale. The pulmonic valves, though neither incompetent nor stenosed, were imperfectly differentiated, and the pulmonary artery was enormously dilated. The ductus arteriosus was obliterated. No organic cause was found for the dysphagia which was such a prominent feature of the case. He suggested that the congenital malformation and the inco-ordination of swallowing were both due to a defective development of the central nervous system, probably of the medulla, and compared a case described in the 'Revue des Malades de l'Enfance' for December, 1903, by P. Hansallin and L. Richon. (*Exhibited January the 15th*, 1904.)

Discussion.

The Chairman (Dr. Porter Parkinson) asked how far the dilatation extended along the pulmonary vessels, and whether the small vessels in the lung were also dilated.

Dr. Eric Pritchard replied that, as far as he could examine, they were all enlarged throughout. There was slight collapse, and a little emphysema. The broncho-pneumonia only came on towards the end. There was no reason to suspect an impeded circulation in the lung.

* *Vide* Case No. 12, on p. 38 in the present volume.

31. SPECIMENS OF CONGENITAL HYDRONEPHROSIS, WITH DILATATION OF THE URETERS AND HYPERTROPHY OF THE BLADDER.

By J. M. FORTESCUE-BRICKDALE, M.D.

In the first specimen both ureters were markedly dilated and tortuous-looking, about the size of the small intestine. Both kidneys were hydronephrotic and microscopically showed much cirrhosis and dilatation of the tubules. The bladder walls were about 5 mm. thick, and at the junction of the bladder and the urethra were two small crescentic folds of mucous membrane, which, however, were not large enough to obstruct the passage, as a catheter passed easily post mortem. The specimen was taken from a child, aged 1 year, who died at the Children's Hospital, Bristol, of wasting and broncho-pneumonia, under the care of Dr. Rogers. There were no symptoms of urinary obstruction during life, and no phimosis or abnormality of the urethra. The second specimen was less marked, the right ureter only being noticeably dilated. There was also some hypertrophy of the bladder, and microscopically the kidney showed similar changes to those in the first specimen, though in less degree. There was no obvious obstruction in the urethra, but the ureter was much narrowed as it entered the bladder. This was taken from a child aged five months who died in the Children's Hospital, Bristol, of suppurating spina bifida, associated with a large hydrocephalus, under the care of Dr. Elliott. Holt* states that other abnormalities are not usually associated with these cases. In neither case was there any arterial degeneration such as is described in Bryant and Hale White's case†.

(*Exhibited January the 15th*, 1904.)

* 'Diseases of Infancy and Childhood.'

† 'Guy's Hospital Reports,' vol. lv, p. 17.

Discussion.

Mr. Thomson Walker said it was difficult to give an opinion on preserved specimens such as these, but in the larger there seemed to be a partial membranous septum at the junction of the membranous with the prostatic urethra. Obstruction in such cases need not be absolute; comparative obstruction of urine was followed by hypertrophy of the bladder, and the dilatation of the ureters and kidneys probably resulted more from the obstruction caused by the thick bladder wall than from the original urethral obstruction.

Dr. Fortescue-Brickdale (Bristol), in reply, said with regard to the slight obstruction apparently present in the urethra, he did not observe it when he first opened the urethra, but he did when he put the specimen up, and he thought it was artificial.

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32. TWO SPECIMENS OF TUBERCULOUS ULCERATION OF THE STOMACH.

By J. M. FORTESCUE-BRICKDALE, M.D.

The first specimen was from a child aged 2 years who died of general tuberculosis at the Children's Hospital, Bristol, under the care of Dr. Elliott. The small intestine was also extensively ulcerated, the abdominal and thoracic glands were caseous, and there were miliary tubercles in the pleuræ, lungs, liver, and spleen. The gastric lesions consisted of several round ulcers, about 1 mm. in diameter, with thickened edges, situated on the posterior wall near the cardiac end of the viscus, and also two irregularly shaped ulcers, of larger size, one on the posterior wall near the pylorus, and the other at about the middle of the greater curvature. There were miliary tubercles on the peritoneal surfaces of the ulcers. In the second specimen there were numerous small round ulcers similar to those in the first case, measuring about 3 mm. in diameter, and mostly situated at the cardiac end. Two were seen close together, separated only by a narrow bridge of mucous membrane, which suggested that the large ulcers in the first specimen might have arisen by coalescence of

several smaller ones. Miliary tubercles were also seen on the peritoneal bases of these ulcers.

In neither case were any symptoms pointing to gastric ulceration observed during life, and the search for tubercle bacilli in two ulcers which were excised post mortem was not successful. It is somewhat remarkable that in both cases the primary tuberculous lesion appeared to be in the mesenteric glands, as these were much larger and showed more fibrous tissue matting them together than any other of the affected glands. Hamilton* states that only fifteen undoubted and nine probable cases of tuberculous ulcer of the stomach have been recorded.

(*Exhibited January the 15th*, 1904).

Discussion.

Dr. G. A. Sutherland asked whether, in the case of tuberculous ulcer of the stomach, there had been any symptoms during life.

Dr. George Carpenter had made a large number of autopsies in general tuberculosis, but he had met with but two examples of the condition, and in neither case were there symptoms during life suggestive of such a complication. The ulcers in his cases were quite numerous, small, round, with thickened edges, and miliary tubercles on their peritoneal surfaces—in fact, quite like the specimen exhibited by Dr. Fortescue-Brickdale. In both Dr. Carpenter's cases there was general tuberculosis. As a possible occurrence in tuberculosis, the condition was of interest from a pathological point of view, though it did not appear to be of much moment from the clinical side.

Dr. Fortescue-Brickdale (Bristol), in reply, said there were no symptoms relating to the stomach during life.

33. CURIOUS CONGENITAL CYSTS OF THE LUNG ASSOCIATED WITH DEXTROCARDIA CAUSED BY PRESSURE FROM THE CYSTIC LUNG.

By GEORGE CARPENTER, M.D.

Rosina P—, aged five months, attended the out-patient department of the North-Eastern Hospital on November

* 'Johns Hopkins Hospital Bull.,' April, 1897.

the 16th, 1903. She had been ill a fortnight with "wheezing at the chest," but before that she was quite well. She was breast-fed. The first child was stillborn; the next died at five weeks old; the child following also died. The fourth and fifth were alive and well, the sixth had fits, the seventh was well, and the eighth was the patient.

On examination the child was pale, with a cyanotic tint, and there were signs of bronchitis.

The *cardiac impulse*, which was weak, was in the fifth interspace, half an inch outside the nipple line on the *right side*. The superficial *area of cardiac dulness* commenced at the fourth right costal cartilage. The spleen was one finger in front of the costal margin and the liver two fingers below the costal margin in the nipple line.

The infant was ordered a cough mixture and improved, but on November the 27th it was admitted into the hospital in a collapsed condition, with a temperature of 97° F. On its recovery from the collapse the temperature ran up to 103·5° F., the respirations were 84 to the minute, and the pulse-rate 144 to the minute. It became convulsed and died on the 29th.

At the post-mortem examination the heart was found as indicated during life. It was *not* rotated on its axis; it was thrust over bodily to the right side. Its valves and blood-vessels were normal. The right *lung* was bronchopneumonic. The lower lobe of the left *lung* was slightly adherent to the chest, and it appeared to be converted into an airbag. The other organs were quite healthy.

The specimen was exhibited as it was removed from the body, so as not to destroy the naked-eye appearance found at the autopsy. On further examination almost the whole of the lower lobe was found to be converted into a cyst the size of a small orange (Fig. 1). It contained nothing but air. It was divided into four or five secondary smaller cavities by septa which were perforated by circular apertures. The walls of the cyst were lined with a yellowish granular material, the naked eye appear-

ance of which presented nothing very definite. The bronchus traced down was found to enter a small area of comparatively unaffected lung. None of the divisions of

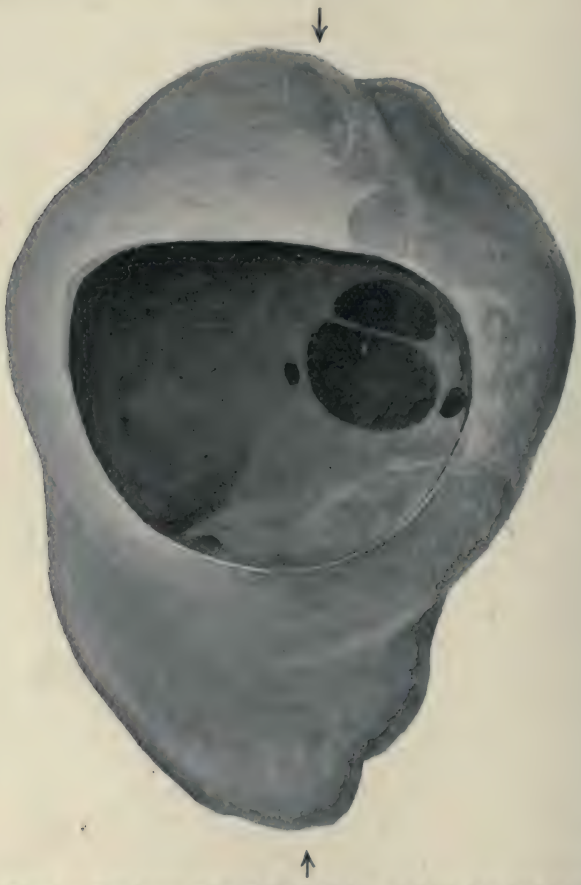

FIG. 1.—The lower lobe of the left lung, which has had a part of the cyst-wall removed so as to display the large cyst and perforations in it which lead into another and smaller cyst. The arrows denote the line of section which was subsequently made in this specimen and from one side of which Fig. 2 has been drawn.

the bronchi could be found to enter the cyst. Below the cyst was another portion of apparently healthy lung,

which on being cut into was found to be composed of numerous small cysts of various sizes, some of which contained granular material, and some were empty (Fig. 2).

Had it been a single cyst one might have thought it

Fig. 2.—Fig. 1 has been cut into two at the part shown by the arrows. Two large cysts and several small cysts, together with still smaller cysts, have been brought in view by this operation, and are here shown.

a sequence of some destruction of the lung, but the presence of numerous cysts seems to suggest that the condition is more of the nature of congenital cysts, akin to those of the liver or the kidney.

(*Exhibited January the* 15*th*, 1904.)

Discussion.

Dr. E. P. Baumann asked what were the contents of the cysts. He once performed a post-mortem on a child who died of empyema, and in the lung on the affected side were two cysts, and they were filled with pus. It was thought in that case that the cystic change was secondary to the empyema.

Mr. A. H. Tubby expressed the hope that a drawing of the specimen would be made, to be included in the 'Reports.'

Dr. George Carpenter, in reply, said that pricking the sac caused instantaneous collapse of its wall. The condition was not diagnosed during life; there were no symptoms. Apparently there were ordinary breath-sounds over that side. He had not found any trace of pus in the cysts. It seemed to him to be a case of congenital cystic lung, and there was no suspicion of an empyema.

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34. THE RELATION OF CERTAIN EXTRA- AND INTRA-CRANIAL HÆMORRHAGES IN THE NEWBORN.

By J. HOWELL EVANS, M.Ch.

In connection with extra-cranial hæmorrhages—cephal-hæmatomata externa—which are occasionally met with, and in connection with intra-cranial hæmorrhages, namely, those extra-dural or cephalhæmatomata interna which we rarely see, and the meningeal or cortical hæmorrhages the destructive influences of which are so frequently encountered, a common explanation has not, as far as I am aware, been advanced.

Such hæmorrhages occur irrespective of many factors which have long been considered as the causes of their production; they arise irrespective of the lie and position of the fœtus in utero, without any relation to a breech or vertex presentation, and in labours unaided by forceps, as well as in labours in which such mechanical assistance is employed. Lastly, even a difficult adaptation of the fœtal head to the maternal pelvis is oftentimes absent.

The injury is done during the neonatal transit, but the blood is effused during the reactionary period which follows, inasmuch as when the extra-uterine life supervenes upon the antenatal, very profound and important changes follow in the circulatory system, in the blood-pressure, and even in the blood itself: dependent upon such changes these hæmorrhages appear.

The fœtal head is relatively large, and in its adaptation to the maternal pelvis its contour is markedly altered.

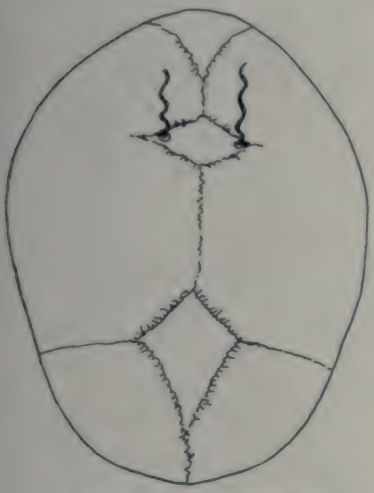

FIG. 3.—Sagittal fontanelle (after Laplace).

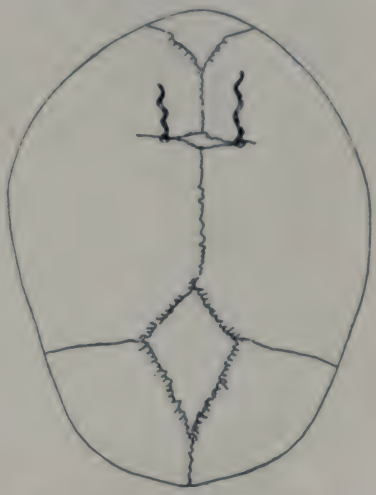

FIG. 4.—Sagittal fontanelle.

This moulding of the head is due to the overlapping of the bones at the sutures; one parietal overrides the other at the sagittal suture, while at the coronal and lambdoid sutures both parietal bones override the frontal and occipital bones.

If this overlapping is excessive, or if, owing to variations from the normal ossification of the bones of the cranial vault, the necessary moulding occurs more particularly at certain accessory sutures, then the process becomes pathological.

It is in relation to these latter (accessory sutures) that

I desire to elucidate the production of these hæmorrhages (Figs. 3 and 4).

I have, therefore, sought some common factor which may act whether the moulding of the head take place from the superior cephalic planes or from the inferior cephalic planes; and in the limited space at my disposal I purpose only to consider those hæmorrhages connected with the parietal bone.

The parietal bone is generally developed from one

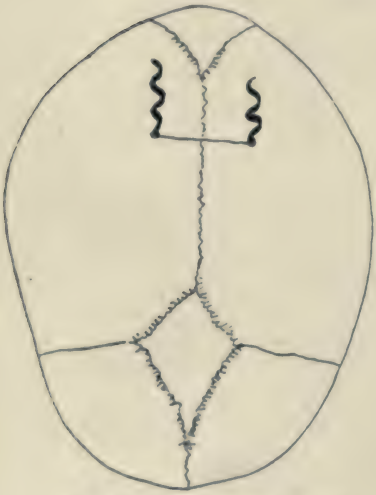

FIG. 5.—Intra-parietal suture.

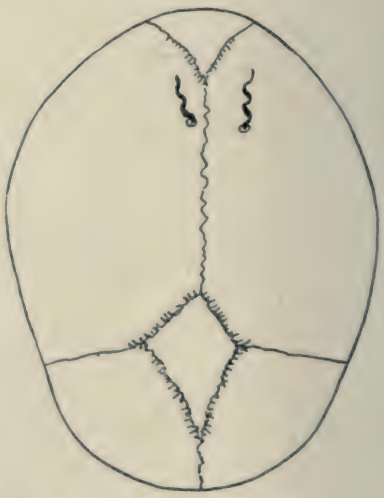

FIG. 6.—Parietal foramina.

centre, whence radiating osteogenetic fibres pass towards the borders of the bone. If, however, the parietal bone become developed from two ossific centres, then from these two centres the radiating osteogenetic fibres pass to the borders of the bone, and at the points furthest from the centres tend to leave gaps. Such a gap exists for a long time in the region of the parietal fontanelle, forming a membranous space. This usually becomes closed about the fourth month of intra-uterine life. It may, however, remain until birth, embarrassing the accoucheur, and a trace of it may even persist throughout life, as the

parietal foramen, serving for the transmission of certain vessels (Figs. 5 and 6).

The presence of parietal foramina is an anatomical fact long known; the theory that such foramina are connected with the blood-supply of the pineal eye has been on all sides accepted; but the importance of these foramina and their transmitted vessels in the production of these various pathological hæmorrhages has scarcely been entertained.

Transmitted through the parietal foramen, intra-parietal suture, or sagittal fontanelle, as the condition may be, are blood-vessels—an artery and a vein—which form one

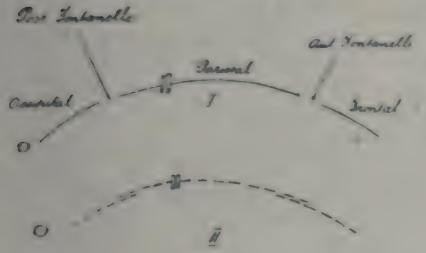

FIG. 7.

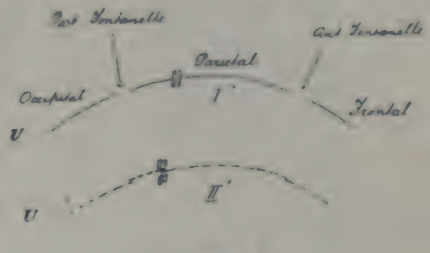

FIG. 8.

Diagram illustrating the moulding of the vault of the fœtal skull in the antero-posterior plane during its transit through the birth canal. O. Ordinary, vessels escape. U. Unusual, vessels injured. I, I'. Before moulding. II, II'. During moulding.

of those important channels of vascular communication between the structures which lie without and those which lie within the skull.

In a fœtal skull of normal ossification subjected to normal and even to excessive moulding these vessels are safe from injury, but with accessory sutures and irregular moulding they are liable to mechanical injury. This injury may lead to—Rupture of the artery (*a*) on outer side of skull; (*b*) on inner side of skull; tearing of the vein; laceration of both vessels; thrombosis of these vessels (Figs. 7 and 8).

Moreover, such an injury may occur on both sides,

but is more liable to be found in connection with the parietal bone which lies outermost. To these forms of injury all vertical hæmorrhages of the reactionary period of the new-born can be traced.

I have been able to produce the external cephalhæmatomata artificially by mechanical pressure, also by lacerating or dividing the vessels with a tenotome, and to demonstrate the same by rhythmical injection of the ruptured vessels with a coloured preparation.

(*Read January the 15th*, 1904.)

Discussion.

Dr. G. A. Sutherland said the Society was very much indebted to Mr. Evans for bringing the paper and demonstration forward. It bore upon a subject in which both surgeons and physicians were very much interested; surgeons had to deal with external cephalhæmatoma, and physicians had to do with cases of birth palsy, which were believed to be due to intra-cranial hæmorrhage at birth. There had been a distinct hiatus in the explanation of the latter conditions, because they were often attributed to cerebral pressure during the process of parturition, and they were described as occurring both in very prolonged and in rapid labours. But when investigating the matter it was found that they also occurred in absolutely normal labours, and without any apparent difficulty whatever. Another theory was that such cases were due to some lack of development in certain parts of the cerebral cortex. The paper of Mr. Evans had suggested another explanation, which was extremely plausible. Still, there was a good deal of theory about his explanation, because clinically he had not been able to present very marked evidence of the condition. Possibly it was difficult to find definite clinical confirmation of his theory. Could Mr. Evans produce post-mortem cases in which he had found a condition of actual hæmorrhage or thrombosis in any given artery or vein? Although he had the greatest respect for Mr. Howell Evans's researches, he was inclined to suspend his judgment as to the explanation given. But if the author could push his investigations somewhat further and convince the profession as to his views, he would be conferring a great benefit upon physicians and surgeons alike.

Dr. C. O. Hawthorne said his sympathies were very much with the remarks of Dr. Sutherland. One felt that the limitations of time had prevented Mr. Howell Evans doing justice to his contribution. It seemed to him that any theory which was

to account for these cases must take cognisance of the fact that one met with paralyses presumably due to hæmorrhages in cases which had not been subjected to any undue pressure during birth—cases of children born in the seventh month, and without mechanical difficulty. He very much appreciated the suggestions offered by the author and the way in which they had been brought forward.

Mr. Howell Evans, in reply, admitted that it was the limitation of time which prevented his elaboration of the subject in the way he would have wished. The work had been steadily carried on by him during four and a half years, and the manuscript of it extended to many hundreds of pages. In dealing with those cases one had to think of the whole variations in the structure of the vessels of the infant, the peculiarities in the anatomical distribution, between the veins which entered at the superior longitudinal sinus and those which ran from the pia mater into the superior longitudinal sinus. It would be some months before the long paper he had referred to took form, but he thought it would be seen that there was scarcely an incident in such cases which was not accounted for by his explanation. With regard to undue pressure, that was the point upon which he had been trying to lay particular stress. It was clear how the radiating osteogenetic fibres of the parietal bone went, and it was only necessary for one to be displaced beyond a certain degree from its normal plane to insure rupture, not necessarily of the main vessel, but of any radical of the artery or vein. The absence of post-mortem evidence constituted the chief difficulty in such cases. Those hæmorrhages occurred during the reactionary period from the shock which the infant had received in its transit through the birth canal. The alterations in the blood pressure came on gradually, and became more and more marked as the alterations of the blood circuit through the lungs and heart were changing, until the blood pressure about the third day began to show itself markedly increased. It was on the third day that one would see the true cephalhæmatoma; at any rate, one must give them forty-eight hours. A small hæmorrhage or injury would be followed by Nature's attempt to repair it by means of a clot. The infant's blood, however, was so deficient in the elements which permitted of true clotting that when the blood pressure rose a little the small clot was dislodged. On the outer side it was limited by the attachments of the dura mater to the bone, and inside, when it was extra-dural, it was limited in a similar way. But when it got to the meninges or the cortex it was not limited, and it became diffuse owing to its fluidity; thus the extensive cortical hæmorrhages arose. He had carried out experiments the results of which supported the theory he had advanced. With regard to the difficulty in presenting post-mortem evidence, if the fœtal head be subjected to very severe

pressure two things occur – marked trauma transmitted to the basal ganglia and basal hæmorrhages—which generally lead to death. If the pressure is less severe the child may lie between life and death for several days, finally recovering. During this period of uncertainty the blood pressure is too low to produce a hæmorrhage, and by the time this pressure has become markedly increased the intra-vascular clot has become firm enough to withstand it. Lastly, the presence of an external cephalhæmatoma is undoubtedly sufficient evidence of the vigour of the infant, and an indication that it will survive.

35. A CASE OF ENDOTHELIOMA OF THE BRAIN, WITH CHRONIC HYDROCEPHALUS.

By W. C. CHAFFEY, M.D.

In the 'Reports' of the Society for the Session 1902—1903, on pages 257—258, a short clinical note of this case was given, under the heading of "Anorexia Nervosa," together with a photograph of the lad (Fig. 9) during the latter part of his stay in the Children's Hospital, Brighton, where he was for some months under my care.

The following history, obtained from the mother, will be of interest, as it seems to illustrate the fact that serious conditions may sometimes exist within the cranial cavity for many years without giving rise to any definite symptoms of cerebral disease.

E. S—, aged 10 years, had been irritable and sensitive to noises for several years past. At eighteen months of age he frequently had severe screaming attacks, during which he became rigid. He was always thin. About eighteen months ago he often complained to his mother that he could not see clearly. About fifteen months ago he returned from school complaining that he had been hurt by some boys pushing him against the wall of the school playground. Shortly after this happened he began to refuse his food and say he could not swallow. He did not vomit or complain of pain in the head till February

of last year, when the mother brought him to the Children's Hospital. He was admitted and kept under observation, but nothing definitely pointing to cerebral disease could be discovered. He was very taciturn, apathetic, and dis-

FIG. 9.

inclined to associate with the other patients. His eyelids blinked when he was spoken to. There was no squint or paralysis of any parts, and sensation, hearing, and smell seemed to be normal. His pupils were somewhat dilated, equal, and reacted normally to light. The knee-jerks were not obtained. Great wasting of all parts of the

body was a marked feature, and this increased slowly but surely, so that he became much emaciated and scarcely able to move about the ward. His gait, however, was not peculiar. There was no retraction of the head, which was about normal in size. There was no tache cérébrale, and he complained of no headache at that time. No Babinski sign could be elicited, and there were no contractions of the extremities. He persistently refused all solid food, but would drink about three pints of liquid, chiefly milk, per diem.

At that time, owing to the absence of definite cerebral symptoms, the case was viewed as one of hysteria, probably the result of shock, consequent on the loss of his sister, who had died recently.

As he seemed brighter and was able to walk better, he was allowed to return home. Here he was well looked after in every respect, but two months later, October, 1903, he began to develop bed-sores, had attacks of pain in the head with screaming, almost constant tonic contractures of the extremities, especially on the left side, and passed his urine and fæces involuntarily. His discs at that time showed fairly well-marked optic neuritis. It became apparent that he was suffering from increased intra-cranial pressure, due possibly to some tumour pressing on the iter and so causing hydrocephalus. I thought probably he had a growth in the middle lobe of the cerebellum. The patient gradually became unconscious and died.

The post-mortem examination, which I made in conjunction with Dr. Akers, who had charge of the case after leaving the hospital, revealed the following particulars:— *Body generally:* Much emaciated. *Calvarium:* Somewhat more expansive than normal, and the bones rather harder than usual. *Cerebrum:* Convolutions over surfaces of hemispheres somewhat flattened. Chronic thickening and opalescence of pia mater and arachnoid at summit of transverse fissure, obscuring the quadrigeminate bodies and probably constricting the iter. The same condition

affected the ependyma lining the iter and ventricles everywhere. The latter were considerably dilated, and contained an excess of nearly clear fluid, no recent lymph, and no meningitis of recent date; and no tubercles were viewed. The corpus callosum was much softened. Closely adjacent to the posterior aspect of the right optic thalamus and easily detached therefrom was a small brain-like mass, nearly spherical, about the size of a small walnut. This on section presented a pinkish-grey striated appearance, resembling that of many forms of glioma.

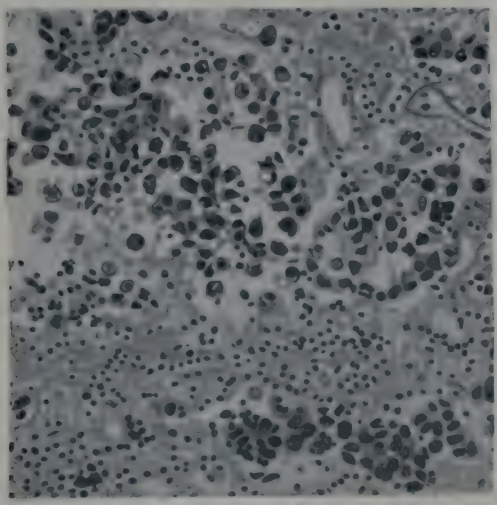

FIG. 10. × 200.

A microscopic examination of this tumour was made by Dr. Targett, of the Clinical Research Association, who reported as follows:—

"The exact nature of this tumour is doubtful, though it is neither a gumma nor a glioma. It will be seen to consist of small epithelial cells arranged in elongated spaces, which are cut in various planes. These spaces appear to be chinks in an abundant fibrillated stroma, not a truly alveolated structure as in carcinoma (Fig. 10). The site of the tumour suggests an origin from the epen-

dyma, and from its structure and probable origin I am disposed to regard it as an endothelioma."

On Dr. Targett's suggestion, I referred the section to Dr. F. W. Mott for confirmation. This I received in due course, so the nature of this rare neoplasm can scarcely be doubted.

Remarks.—I would suggest that the chronic meningitis dated from infancy and was probably tuberculous, though no tuberculous glands were found anywhere. But two small, shotty, yellowish nodules were discovered near the surface of the right lung. It seems to me probable that the meningitis becoming arrested in infancy, the chronic hydrocephalus gradually supervened. I would further suggest that this increased as the result of the blow received in the school playground about five months before he was admitted into hospital, and that the endothelioma, which seemed to be of recent growth, was incited through the same accident.

(*Read January the 15th*, 1904.)

Discussion.

The Chairman (**Dr. Porter Parkinson**) thanked Dr. Chaffey heartily in the name of the Society for coming from Brighton to give the sequel of the case which so many members saw when at that town. The object of the Society could not be better served than by showing morbid specimens, and especially by furnishing the sequel of the cases which had been previously shown.

Dr. George Carpenter joined in the thanks to Dr. Chaffey. His own impression on seeing the boy was that it was a case of anorexia nervosa, and it called to his mind a picture in 'Pediatrics' which appeared some years ago, the patient being hysterical. In that case no unfortunate sequel took place, for the child got well by anti-hysterical treatment. He could not find any evidence of cerebral disease in the boy at Brighton. Dr. Chaffey and Mr. Tubby had spoken on the present occasion about traumatism in inducing new growths. He thought there was a relation also to other diseases. Children would develop tuberculous meningitis after injury, and it was not unusual to find pneumonia and pleurisy following trauma in the same way. He supposed it was fair to assume that they did occur in that way, that the vitality

of parts was lowered by the injury, and that they thus became a prey to the various micro-organisms which were about. Dr. Chaffey's appeared to be a unique case.

Dr. G. A. Sutherland said he also saw the case at Brighton, and his diagnosis agreed with that of Dr. Carpenter, and it was a shock to him to learn that the case had died of cerebral tumour. There was very little vomiting, and an entire loss of appetite, but not a single localising or general sign of cerebral disease. It was very strange that it should have gone on for several months like that, and then finally died of cerebral tumour. Was it possible that the tumour might not have been the cause of all the symptoms? Possibly the condition was essentially one of general neurasthenia or hysteria, the tumour being simply a superadded condition. There was a total absence of gross cerebral disease.

Dr. W. C. Chaffey (Brighton), in reply, said he had thought the symptoms might have been due to hydrocephalus, because the first symptom which arose was defective sight, which was probably a result of increased pressure in the ventricles. The spastic rigidity of the limbs was very marked towards the termination of the case, and might have been due to thickening of the ependyma. The tumour was only on one side, immediately posterior to the right optic thalamus, and easily detached therefrom. If the contractures were all on the left side, one could understand that the tumour being on the right side might have created them, and possibly also pain, but it would not explain contractures on the other side. Probably the hydrocephalus with the pressure on the iter was sufficient to create the symptoms. But he was prepared to admit that the hysterical symptoms, and the contractures and pain on the left side might have been partly due to the presence of the tumour. It was not in a position to destroy the brain to any extent, and he hesitated to attribute symptoms to tumour in the brain unless that tumour really invaded the brain substance.

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36. A CASE OF CEREBRAL DIPLEGIA WITH CHANGES IN THE FUNDUS.

By ROBERT HUTCHISON, M.D.

Albert W—, aged 1 year and five months. A full-time child; easy labour; mother suffered much from vomiting during pregnancy. The first child of the family

was injured at birth, and died after a week from "lock-jaw." There is a girl six years old, and a baby younger than the patient, and both are quite healthy. There is no history of snuffles or rash. The present condition has existed since birth.

The child is well nourished, and lies with all the limbs flexed on the trunk in the intra-uterine position. There is extreme spasticity of all the muscles, the deep reflexes being much exaggerated. The muscles are not wasted. The expression of the child is vacant, and there is apparently complete blindness. The circumference of the head is rather above than below the average. The child is apparently completely imbecile, and is for the most part apathetic, but occasionally gives vent to a peculiar and distressing cry. The viscera are healthy.

Mr. W. T. Lister, who kindly examined the eyes, reports that "the pupils are inactive to light. There are diffuse opacities in the lenses, chiefly posterior-polar. The fundi exhibit extensive choroido-retinitis, and the discs are apparently atrophic."

It is suggested that the case is one of arrested development of the cortical cells, possibly of syphilitic origin.

(*Exhibited February the 19th*, 1904.)

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37. A CASE OF PROGRESSIVE CEREBRAL DEGENERATION (FAMILY TYPE).

By ROBERT HUTCHISON, M.D.

Henry B—, aged 4 years, was healthy till ten months ago, when he began to suffer from fits, which continued off and on for some weeks. Two months ago he "went off his legs," and staggered on trying to walk. He now exhibits some spasticity of the legs with defective co-ordination, and seems to be becoming mentally apathetic. The optic discs are becoming rather pale, and seem to be

passing into a condition of atrophy. There is nothing further of note in the patient's condition.

He was born at the full time, and the labour was easy. There is no history of still-births or miscarriages, but he is said to have had "snuffles."

There is only one other child in the family, an elder brother of six years old, who, when about four, became affected in the same way as this patient, and is now under the care of Dr. Batten. He is completely imbecile.

These cases probably belong to the group of family cerebral degenerations followed by optic atrophy, a few of which have been described.

(*Exhibited February the 19th*, 1904.)

Discussion.

Mr. Sydney Stephenson said with regard to the two cases exhibited, they seemed to belong to a group which had been more or less worked out lately, one in which systemic symptoms, on the one hand, were correlated with fundus changes, on the other. The systemic symptoms were those of cerebral degeneration, and they might vary from mere irritability to idiocy. The eye changes seemed to include pallor, but not necessarily atrophy, of the papilla, smallness of retinal vessels, and fine pigmentary changes in the central region of the fundus. The first case of the kind was described by Mr. Rayner Batten in a child of syphilitic parents, but the child showed fundal changes, and not to any marked extent mental changes. Mr. Donald Gunn and Dr. Still showed a case at the Ophthalmological Society a few years ago which seemed to belong to that class. Dr. R. B. Batten showed two cases last year in which the fundus changes coincided with the mental changes; and a few days ago, at the Ophthalmological Society, Mr. Stephen Mayou showed three members of one family who presented both fundus and mental changes. He, Mr. Stephenson, had seen one case in a girl, aged 12 years, the offspring of a consanguineous marriage. He thought one might suggest that cases of that class belonged to the family eye diseases, of which a certain number had been recently worked out. So-called "nodular" or "lattice-like" corneal changes was one class; pigmentary retinitis was another; and an amaurotic family idiocy (Sach's chronic) was another. Two points seemed to stand out in the ætiology: one was the existence of syphilis in the parents or in the child itself, and the other was consanguineous marriage between the parents.

Mr. Jackson Clarke said these cases most closely resembled

the ordinary spastic diplegias, and for some of the latter surgery could do a great deal in the direction of permanent help to the individual. But in the present cases nothing could be done surgically. It showed the importance of having a thorough examination made by a physician before any definite prognosis or treatment was undertaken in young children with general spasticity.

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38. CASE OF SYMMETRICAL BILATERAL BRANCHIAL FISTULÆ WITH SYMMETRICAL BILATERAL HELICAL FISTULÆ AND MIS-SHAPEN AURAL PINNÆ.

By J. HOWELL EVANS, M.Ch.

Beatrice S—, aged 3½ years, the youngest of three children, came under my observation some three months ago. When the child is viewed in full-face the very large epicanthal folds passing outwards from the broadened bridge of a flat nose become most apparent, but a side-view brings into evidence large flap-like and thin pinnæ, resembling in texture the thin ears of bats and monkeys.

On these pinnæ there are situated symmetrically, at a point corresponding to the site of insertion of the attollens aurem muscle, and consequently looking forward, upward, and outward, fistulæ of the diameter of 2 to 3 mm. and 1 mm. deep.

On either side of the neck (in the same horizontal plane), situated at a point 3·25 cm. from the middle line, at about the level of the cricoid cartilage, is a small red point, the centre of which is drawn in; the sides merge gradually into healthy skin.

Whenever the act of deglutition is carried out these orifices become more drawn in, corresponding much to the aspect of funnel-shaped and deep-reaching cicatrices, which is indicative of attachment to somewhat firm but deep structures. From these cutaneous orifices linear tracts can be felt passing upwards and backwards. These orifices have been closed at varying intervals with the

formation of small cysts, which after a certain degree of intracystic pressure have again opened.

In the cases recorded by Sir James Paget* there was a very definite hereditary influence.

The interest of this case lies in the associated occurrence of helical symmetrical fistulæ, lateral cervical symmetrical fistulæ, mis-shapen pinnæ, and marked epicanthal folds; also in the fact that there was a mandibular tubercle in the mother, preauricular tubercles in the aunt, a nævoid hairy patch in the sister, a small tubercle in the brother, and a minute naso-labial tubercle in the grandmother.

(*Exhibited January the 19th*, 1904.)

Discussion.

Mr. Lockhart Mummery said the case was very interesting, but he did not agree with Mr. Evans that the fistulæ in this case were in the position of the fourth cleft; he thought it was the third. The fourth cleft fistulæ as a rule opened above the sternal end of the clavicle, and fistulæ which opened opposite the cricoid, as the present one did, should be classed as the third cleft. The greatest interest centred round the fistulæ in front of the ear. These Mr. Bland-Sutton had described as due to non-fusion of two of the primary tubercles of the pinna. But there were facts which militated against that view, and there were cases on record in which fistulæ in front of the ear were at least ½ an inch deep, and in one case recorded by M. Lannelongue the fistula was situated in the ordinary place at the commencement of the antihelix and went down to a small cyst with a canal ½ an inch long. He thought they represented the posterior end of the inter-maxillary cleft, and there were several cases which lent support to that view; they should be considered as the homologue of the spiracle of fishes.

The Chairman (Mr. R. Clement Lucas) said the cases showed the extreme hereditary tendency there was. In this child there was evidence of lack of higher development in the condition of the ear, and also in the condition of the fold above the eye on the inner side of the orbit, which he believed was generally recognised by mental experts as a very grave indication of bad mental development. He understood from Mr. Evans that the fistulæ in the neck were closed at first and afterwards burst through. He had seen them mistaken for glands in the neck. He was asked about one case by an insurance medical officer as to whether the case was one of tuberculous gland, the man's policy having been refused on former occasions because

* 'Med.-Chir. Trans.,' 1877, vol. lxi.

the condition was considered to be glandular. It was clearly a cyst in association with the branchial cleft. He had also seen the mandibular tubercles without any clefts, and had traced other deformities in association therewith. Another condition often associated with mandibular tubercle was a line running from the angle of the mouth along the cheek up to the tubercle, indicating that in such a case there might have been lack of closure from the tubercle to the angle of the mouth, and a large oral orifice in consequence. The association of the tubercles with the fistulæ was so well worked out in the present case by Mr. Evans through several generations that he hoped members would bring before the Society similar cases which they might meet with.

Mr. Howell Evans, in reply, said he thought Mr. Mummery was correct (from the present clinical knowledge of such cases) in his remarks about the third cleft; it was the third as usually looked at, but having devoted a good deal of time to the matter and studied the embryological work of Hostenacki, he was inclined to look differently at it. Another reason why he differed was because he viewed it as a reversionary atavism, in its relation to the branchiæ of the tadpole. With regard to the helical fistula, Mr. Mummery referred to the case which had been recorded of a sinus $\frac{3}{4}$ths of an inch long leading down to a dermoid cyst. That instance had upset the view put forward by Paget that the anterior part of those fistulæ corresponded to the outer opening of the first cleft. The probability was that such fistulæ arose between the two primary tubercles found upon the mandibular cleft which entered into the anterior and superior part of the ear. There was still another way of explaining it, viz., that it was reversionary atavism to the type of the ear which was found in all animals with thin ears; such often presented a small sinus associated with the part between the attollens aurem and the retrahens aurem, and it was this depression down which the epiblast had extended.

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39. GASTROTOMY FOR THE REMOVAL OF A PORTION OF A COIN-CATCHER FROM THE LOWER END OF THE ŒSOPHAGUS OF A CHILD AGED 5 YEARS.

By DONALD ARMOUR, M.B.

J. H—, a boy aged 5 years, was brought to the West London Hospital, having swallowed a coin. With the fluorescent screen the coin was seen lodged at the sinus

pyriformis. The house-surgeon passed a coin-catcher down and engaged the coin, but on traction being made to withdraw it the coin-catcher broke off at the junction of the metal part with the whalebone handle. The portion of the coin-catcher (five inches in length) passed down and lodged in the lower end of the œsophagus. Under chloroform the coin was easily extracted through the mouth with a pair of crocodile forceps. The abdomen was then opened to the left of the middle line, and the stomach palpated and found empty. The anterior wall of the stomach was then invaginated, and the finger passed up through the cardiac orifice. The lower end of the coin-catcher could just be reached, but disappeared at once owing to the strong contraction of the cardiac orifice. A small vertical opening was then made in the anterior wall of the stomach, midway between its borders. The finger was again passed through the cardiac orifice and the coin-catcher felt. A pair of long crocodile forceps was passed along the finger, the coin-catcher seized and withdrawn. The stomach wound was sewn up with a continuous catgut suture through all the coats, covered in by a continuous Lembert suture of celluloid thread. The abdominal wound was closed by continuous catgut sutures for the different layers, and interrupted silk-worm gut sutures for the skin. No drainage was used. The child made an uninterrupted recovery without any further symptoms. (*Exhibited February the 19th*, 1904.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said he presumed Mr. Armour used a Lembert suture and did not drain.

Mr. Jackson Clarke asked whether Mr. Armour had any experience of the length of time which cellulose sutures took to become absorbed, especially compared with silk.

Mr. Donald Armour replied that Lembert sutures were used, and he did not drain. The sutures were really linen thread covered with celluloid, and he believed they remained unabsorbed the same length of time as silk.

40. A CASE OF MONOCULAR VERTICAL NYSTAGMUS.

By ARNOLD LAWSON, M.D.

The right eye oscillates continuously. The movements of the left are normal. The oscillations are small, very fast, vertical in direction, with a slight wheel-like rotation. They are most marked when the eyes are directed upwards, and least so when directed downwards. The eye is an amblyopic one, with a leucoma of the cornea due to an old perforating ulcer probably contracted from ophthalmia neonatorum in infancy. The eye exhibits a well-marked convergent strabismus, rather lessened in degree by an internal tenotomy a few months ago. The tenotomy seemed at first to have a good effect on the nystagmus, but the condition of the eye in this respect has gone back to what it was before operation. Vertical nystagmus is always rare, and monocular vertical nystagmus exceedingly so. Of the few cases of monocular nystagmus that have been recorded the majority appear to have been horizontal.

(*Exhibited February the 19th*, 1904.)

Discussion.

Dr C. O. Hawthorne said he had notes of at least one case of monocular nystagmus. He understood from Mr. Lawson before he left that in the cases on record, as in the one now shown, the affected eye had been practically blind. The instance he now quoted was a child aged between 2 and 3 years, the subject of alternating convergent strabismus. The fundus, cornea and media were normal. The child was not blind in either eye, though he could not say whether the vision was of normal acuity. It was observed that the nystagmus ceased when the pupil was dilated by atropine.

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41. EARLY FRIEDREICH'S DISEASE.

By J. JACKSON CLARKE, M.B.

Henri B—, aged 4 years. The parents have noticed an increasing weakness in the patient's legs. The knee-

jerks are exaggerated, Babinski's sign is present on both sides. Both feet drop into the talipes equinus position. He walks with the arms held back, the knees bent, and the feet apart.

A brother of the patient, now aged 23, and a cripple, had a similar history to the patient, but his weakness began a little later, at six years. He has now some nystagmus, marked spasticity in both lower limbs, adduction of the thighs, rigid extension of the knees, and rigid flat-foot being present. His disabilities would have been greater had he not been operated on earlier in the course of his disease by Mr. Clutton, who divided the hamstrings and the tendo Achillis on each side.

Is surgical treatment justifiable in Friedreich's disease? In my experience it is, except in the later stages of the disease. The degrees and the rate of development of the spinal lesions appear to differ considerably, so that in some cases the correction of deformity (generally equino-varus) enables a patient to get about comfortably for the rest of a life that is not shortened by the disease. Even where the disease is progressive the correction of deformities enables the patient to enjoy locomotion for several years longer than would otherwise be the case. In the earliest stages all that is required is a light instrument to prevent the foot dropping.

(*Exhibited February the 19th*, 1904.)

Discussion.

Dr. Porter Parkinson said it would be very valuable if the case could be brought for inspection in another year or two. At present he thought there was evidence of a lesion of the lateral columns of the cord, and the gait was somewhat similar to that in Friedreich's disease. There were no ocular signs nor any arm symptoms, and the spine appeared to be normal. Probably the condition would have passed unsuspected were it not that there was a family history of the disease.

42. SECONDARY ANGIOSARCOMA OF THE LUNG IN A CHILD AGED 3 YEARS.

By W. A. WILLS, M.D.,

AND

DOUGLAS DREW, B.S.

A. T—, aged 3 years. This specimen of the thoracic organs is shown as a sequel to the specimen of adeno-myxosarcoma of the left testis, shown by Mr. Drew at the meeting of the Society on November the 12th, 1903.*

In January, 1902, Mr. Drew saw this child at the North-Eastern Hospital with enlargement of the scrotum of three months' duration. After twice tapping a hydrocele in April, 1902, the enlarged left testicle was removed. There was no thickening of the cord or vas deferens, but the testicle was as large as that of a child of ten, and was found to consist microscopically of an adeno-sarcomatous growth, with a cellular stroma which had undergone mucoid degeneration, enclosing cystic spaces.

Mr. Drew saw the case in September, 1903, for swelling of the abdomen, wasting, and cough of a month's duration, but neither he nor Dr. Parkinson, who examined the chest, found any signs of a growth.

On December 9th the child was admitted under Dr. Wills at the North-Eastern Hospital as a case of empyema. The child was very ill, with great dyspnœa, and there was great impairment of movement of the left chest, with wooden dulness all over the left front, axilla and back, extending 2½ inches to the right of the sternum in the third and fourth interspaces, owing to the displacement of the heart. There was bronchial breathing at the left apex in front, but below this all over the dull area the breath sounds were absent. There was exaggerated breathing on the right side.

The physical signs so decidedly suggested fluid that an

* *Vide* No. 11, Case 2, page 36 of the present volume.

operation for draining the supposed empyema was at once performed, but no fluid was found. When Dr. Wills saw the child two days later he again could not believe that fluid was not present, and the wound was again opened up. It was then felt that the lung was extremely hard, and Dr. Wills thought that the hardness was probably due to a new growth; three days later the child died.

On post-mortem examination a large solid growth was seen occupying the whole of the left chest, and extending well beyond the middle line, pushing over the mediastinum and heart. There was no glandular enlargement anywhere, and no secondary growth. The absence of one testicle was not noticed.

After hardening the thoracic organs in formalin for some weeks they have been examined more in detail, and the following is an account of the specimen shown: It consists of the lungs, heart, main vessels, trachea, and bronchi. Practically the whole of the left lung is replaced by a soft new growth, into which there is much blood extravasation, and which has undergone considerable degeneration, owing, no doubt, to rapid growth. The pleura is almost universally adherent, and considerably thickened. The only part of the lung not involved in the growth is a portion of the left upper lobe. The growth has invaded the left auricle, forming a projection the size of half a walnut, pushing the lining membrane before it, but not producing any endocarditis. Where the neoplasm is not degenerated it is white, and mottled with areas of red (hæmorrhages). The mediastinal glands are somewhat enlarged, but those around the root of the left lung are much enlarged, and are continuous with the growth in the lung itself. The glands are fairly firm in consistence.

Microscopical examination of the best preserved parts shows a reticulum formed by epithelioid cells, with large oval nuclei, the cells being very variable in shape and size. In some parts the trabeculæ are so arranged as to present a pseudo-acinous appearance. In other places the tumour is composed of similar epithelioid cells without any definite distribution. In all parts there is much ex-

travasation of blood-corpuscles, and there are present many new and thin-walled vessels. In some places there are tubules, probably lymphatic vessels, crowded with large spheroidal cells. In other places the tubules have an acinous appearance, being lined by epithelioid cells, which on section show a distinctly cuboidal shape.

This child, when admitted on the last occasion, was not known to have been previously under Mr. Drew, and this fact was only ascertained some few days after death.

Although the diagnosis of malignant disease of the lung was made during life, the real interest of the case lies in the fact that it was on the point of being recorded after post-mortem examination as a case of primary sarcoma of the lung, and it was only by chance almost that the lung growth was found to be secondary to a growth in the testicle. If such a mistake can be made in a hospital case, with a careful post-mortem examination, it suggests that the few other recorded cases of primary sarcoma of the lung must be accepted with some caution. Dr. Rolleston has recently recorded the cases of primary sarcoma of the lung which have been described with any certainty, and he finds only eight, of which his own case, aged 13, was far the youngest. But our experience in this case makes one feel that all supposed cases of primary lung sarcoma should be scrutinised with great care before being so described.

(*Exhibited February the 19th*, 1904.)

Discussion.

Mr. Douglas Drew said he owed the Society an explanation, but Dr. Wills had kindly made it for him, as to how the case came to be exhibited in November last as one of cure after removal of sarcoma of the testicle. From the time of the operation in April, 1902, until the time when he read the notes of this case, he had had the child under constant observation, expecting to see a recurrence of the disease. On the last occasion he saw the patient it was somewhat wasted, but he could find no evidence of recurrence. To make doubly certain he asked Dr. Porter Parkinson to examine the chest, who found no evidence of disease, so felt justified in reporting it as a cured sarcoma. In the remarks he made at the time he said he thought the

favourable result was due to the fact that the growth was largely of an adenomatous nature, describing the case as one of adeno-myxosarcoma, on the report furnished by Mr. Targett, of the Clinical Research Society. There was a large epithelial element in it, and papillomatous growths in the spaces. The portion now shown under the microscope resembled the primary growth, but the rest of it was of the nature of an angiosarcoma or endothelioma.

Dr. Porter Parkinson said when he examined the case he did so with the idea that if anything abnormal was to be found it would be due to secondary growth, because Mr. Drew had told him the history of the case, and he had himself seen how frequently there was a connection between testicular growths and those which were intrathoracic. He thought that the explanation was that the growth started at the root of the lung, and that it came to the surface comparatively late, the growth being very rapid.

Dr. W. A. Wills, in reply, said the signs exactly resembled fluid in every way. His own impression was that the growth was extremely rapid, and that it had mainly grown between the time Dr. Parkinson saw the case and that at which he saw it himself. There was considerable hæmorrhage into the tumour, indicating rapidity of growth, and the displacement of the heart was so considerable that it could not have existed for very long.

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43. ANÆSTHESIA AND RECURRENT PRESSURE SORES IN SPINA BIFIDA CASES.

By F. PARKES WEBER, M.D.

Etty G—, now aged 4½ years, the only child of its parents, was born with a ruptured spina bifida of the upper sacral region. This was treated by operation on the following day. Since then the child has had paresis of the lower extremities and persistent complete incontinence of urine and fæces. She generally has pressure sores over the ischial tuberosities, which, however, heal up when she is kept in bed and attended to. During the first year of life the size of the head used to be repeatedly measured at a London hospital, and it is probable that the presence of hydrocephalus was diagnosed, a condition

which has long been known to be occasionally associated with spina bifida. There is no history of any "maternal impression," nor is there any evidence of congenital syphilis, or of malformations in relatives.

At present the child is fairly well nourished and apparently not mentally defective. The forehead is somewhat bulged, but if hydrocephalus was formerly present, it has evidentally not progressed. There is nothing pointing to intracranial disease by ophthalmoscopic examination. At the site of the operation on the spina bifida (level of the first and second sacral vertebræ) there is considerable scarring and thickening of the tissues over the spinal column. The child cannot walk by itself, but when supported it has a spastic cross-legged gait, on its heels, with the thighs kept tight together and the feet turned inwards. The knee-jerks are very excessive. On admission to the hospital, at the commencement of the year, there were unhealthy sores over both ischial tuberosities and on both labia majora. There is certainly complete anæsthesia over these parts, and the ulcers which were present are doubtless to be regarded as "trophic ulcers" as far as bed-sores in spinal diseases can be termed trophic ulcers. For a few days there was fever, but this has disappeared and the ulcers have nearly healed up by keeping the child in bed and applying simple dressings. I am afraid, however, that the sores will reappear when the child leaves the hospital, for this happened after they had been quite healed up on a previous occasion.

I know a boy, Christopher T—, now aged 15 years, with spina bifida of the sacral region, who suffers from exactly similar pressure-sores over the ischial tuberosities. He has anæsthesia limited to the neighbourhood of the sores. From birth he has had incontinence of urine and fæces. There is some degree of spasticity of the lower extremities, with increased knee-jerks and *pes cavus* on both sides, but the muscles of the thighs are well developed and he can walk fairly well. There is no hydrocephalus, and though he is rather small and pale his general develop-

ment is not bad. In this boy's case the anæsthesia, pressure sores and incontinence of fæces and urine were certainly not caused by operation, for no operation was performed on the spina bifida till he was about 6 years old. The pressure sores occasionally heal up, but always break out again after a short time.

(*Exhibited February the 19th*, 1904.)

FIG. 11.—To illustrate pressure sores and spina bifida in the second case.

Discussion.

Mr. Howell Evans said in connection with the ulcers which appeared on the lower parts of children who were suffering from spina bifida, he had seen three cases, one of which Mr. Bernard Pitts had operated upon some years previously to close up the dorsal deficiency as much as possible. Complete incontinence of urine and fæces called for such a procedure, but it was not curative. The child had many ulcers in the buttock which

occasionally healed, again to recur. He only saw the child after it had been operated upon. Another case came occasionally to Great Ormond Street in Mr. Kellock's out-patients, with ulcers on the knees; and a third came under his observation, while he was doing Mr. Kellock's work, in August, 1902, with what appeared to be a lympho-lipomatous condition of the foot—that is to say, fibro-fatty tumours of the foot. There were evidences of an infective process, but a few days later an ulcer appeared on the other foot, and close examination revealed spina bifida occulta. He looked upon such trophic lesions as primarily traumatic, but in those conditions the child's sensibility was diminished and the trauma was not taken account of. Nevertheless such a lesion could not appear without a primary injury in a part of increased vulnerability.

Mr. Douglas Drew said that nutritional changes were frequently met with in connection with spina bifida. He had in mind the case of a child which was cured by injections, one of Mr. Bland-Sutton's cases. The child had grown up, and had been under his, Mr. Drew's, care for three years with some slight deformity of the foot, but that had been improved by tenotomy and dividing the fascia. The limbs were very cold and blue at times, showing that they were not very well nourished. No ill effects followed the tenotomy.

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44. BONE-LIKE CEPHALHÆMATOMA IN AN INFANT.

By GEORGE CARPENTER, M.D.

The patient was a male infant aged 11 weeks, born at full-term, the labour lasting five hours and without instruments. At birth there was a large lump over the skull which remained soft for three weeks afterwards and "felt as if it was full of water." It subsequently became hard.

When brought to the North-Eastern Hospital for Children there was a large boss over the right parietal bone 3 inches in diameter and ¾ths of an inch in height. The boss felt quite hard, but in its centre there was a tiny depression. The highest point of the tumour was at the back. The history was all in favour of a cephalhæmatoma, which it was thought to be; but the physical charac-

teristics just related did not support that view, there being an absence of the peripheral ridge of bone, which, taken in association with a central depression, often simulates a depressed fracture. The induration was very like a cranial osteophyte, and for these reasons it was shown to the Society. To clear up the diagnosis and prove the truth of the original impression it was skiagraphed by Mr. Sale-Barker. The screen showed the line of the bones of the skull running clearly below the tumour, thus excluding the possibility of it consisting of bone. Two skiagrams which were taken confirmed the screen diagnosis. The boss was said to be much smaller than it was formerly.

(*Exhibited February the 19th*, 1904.)

Discussion

The Chairman (Mr. R. Clement Lucas) said hæmatomata might occur in three situations on the scalp—either underneath the skin, or beneath the aponeurosis, or underneath the periosteum. Those which were the hardest were those occurring deepest. And in some cases where hæmorrhage took place underneath the periosteum they became ossified. The skiagram showed that that was not the case in Dr. Carpenter's specimen. Perhaps Dr. Carpenter would say where the hæmorrhage was effused in the case.

Dr. Carpenter, in reply, said the skiagram showed that the effusion had taken place on the surface of the bone. There were no bony elements in the hard tumour.

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45. UNCOMPLICATED MYOCARDITIS IN A GIRL AGED 7 YEARS. ASSOCIATED WITH SIGNS OF CARDIAC FAILURE FOLLOWED BY SUDDEN DEATH.

By GEORGE CARPENTER, M.D.

Ada M—, aged $7\frac{1}{12}$ths years, was brought to the North-Eastern Hospital for Children on January the 11th, 1904. Her health had been quite good until

July, 1903; at that time she had an ulcerated mouth and her breath was very offensive. Since August she had been an out-patient at the Adelaide Infirmary. She went to Sunday School on January the 10th, but fainted while there and was carried home. There was neither history of diphtheria nor rheumatism to be obtained. She had never had scarlet fever. When I saw her she was somewhat cyanosed. Her pulse was weak at the wrist and the first cardiac sound was not good. The second at the left base was accentuated. There was no extension of dulness to the right and there was no regurgitant pulse in the vessels of the neck. A few bronchitic signs were to be heard over the lungs and the free edge of the liver was felt two fingers' breadth below the costal margin in the nipple line. The patellar reflexes could not be obtained; the soft palate acted but not very freely. Her urine was nearly solid with albumen. I thought her to be suffering from myocarditis and admitted her into hospital.

On the day of admission the highest pulse-rate was 136 to the minute and the highest respiration-rate 48 to the minute. Nothing abnormal was detected in her fundus oculi. Her temperature was 97·8° F. The following day she was much worse. She was cyanosed; the breathing was shallow and irregular; the heart sounds were very faint and feeble and the pulse could not be felt at the wrist. She died at 3.30 p.m.

The post mortem was made the following day at 2.30 p.m. She was a well-nourished child; rigor mortis was passing off. There were 4 oz. of serum in the abdominal cavity and 1½ oz. in the pericardial sac. The heart weighed 7 oz. Its valves were healthy. The right side was slightly dilated; it was thin walled, pale and friable. The left ventricle was of normal thickness; it also was very pale and friable. Both auricles were dilated and pale. The heart was pale on its endocardial surface as well as on its pericardial aspect. All its cavities were filled with blood clots. The extreme pallor of the heart was the most striking feature. The lungs were heavy and dropsical; the right weighed 9½ oz., the left 7½ oz. The liver was pale; its

free edge was on a level with the costal margin in the nipple line. The right kidney weighed 3¼ oz., the left 3 oz.; they were pale. The spleen and pancreas were normal; the former weighed 1½ oz., the latter 2 oz. The mucous membrane of the stomach was injected and it contained a little altered blood. The intestines were normal. The brain, which weighed 40½ oz., was normal.

Microscopical examination.—The heart muscle was stained by Marchi's method, which showed fatty degeneration of some of the muscle-fibres of both ventricles and

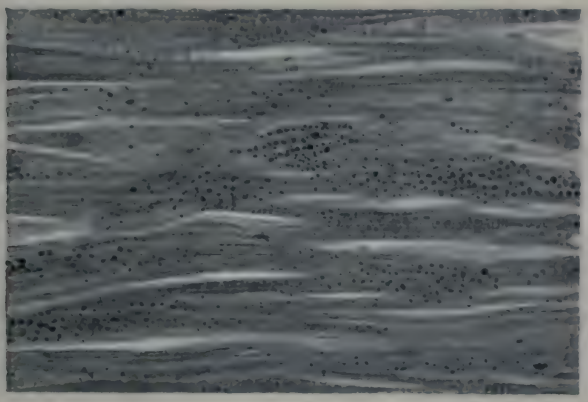

FIG. 12.—Microscopic section of the left ventricle, showing fatty degeneration of the muscle (stained Marchi). Longitudinal striation of the muscle-fibres is pronounced, but cross striation where present is but faintly indicated.

auricles. The accompanying illustration is taken from a part where these changes are well marked. The straight tubules of the kidney contain a considerable number of red blood-corpuscles. The corpuscles are shrivelled and old, they are widely distributed and fill the lumen of many of the straight tubules, forming casts there. In addition the two thirds of the kidney nearest the pelvis contain an amount of scattered pigment granules. These granules are not uniformly distributed, certain areas being quite free from them, while in other parts they are very

numerous and closely aggregated. They vary in colour from golden yellow to nearly black. Some are incorporated with the cells of the tubules but others lie on their surface. In places their origin from the altered blood-cells can be made out.

Remarks.—In Volume III of the 'Reports' (pp. 170—181) I drew attention in this country, under the heading of "Uncomplicated Myocarditis in Children," to the occasional occurrence of clinical examples of primary degeneration of the heart muscle in the young with all the symptoms of valvular disease and without endocarditis, of which the case whose history I have just narrated and from whom the specimens which I now show were taken is an excellent example. Long prior to this, in 1896, I had called attention to the condition * in America.

In my other cases, one, post diphtheritic, whose cardiac symptoms were much like those of the child under discussion, recovered. Another of unknown causation died and interstitial myocarditis was found post mortem. A third, of rheumatic origin, died suddenly and fatty degeneration of the heart muscle was found. A fourth, a boy of 12 months, showed extensive fibroid disease of the heart. The child died suddenly and general tuberculosis was found at the autopsy. The cause of the heart disease was not obvious and possibly rheumatism was responsible. There was no evidence of syphilis. In the present example the cause for the fatty degeneration of the heart is not certain, but I am inclined to the view that it is post diphtheritic; at least, that appears to me the most likely solution.

I claim for myocarditis, including in this term cases of fatty degeneration of the muscle fibres pure and simple, a distinct clinical entity. I have used the term "myocarditis" with intent, and not because I wish it to be understood that I consider there is anything about the condition of an inflammatory nature. For one reason it is customary

* "Two Fatal Cases of Heart Disease in which the Cardiac Muscle was alone Involved," 'Pediatrics,' vol. ii, pp. 234—238.

at the present time in text-books on children's diseases to group such conditions under the heading "Myocarditis"; but I have used the term chiefly because I wish the case to appear as an addendum to my recent communication on "Myocarditis in Children."

The disorder in a pronounced case, in the absence of complaints known to produce it, can be diagnosed during life; and it is a state, dangerous though it be, from which recovery can and does take place, even in a severe case, as I have more than once observed. The incidence of cardiac symptoms in association with diphtheria, rheumatism, or sepsis facilitates the diagnosis. Myocarditis is likely to be overlooked in cases where the primary disorders which produce the complaint have passed unnoticed, as is very likely to occur and as a matter of fact frequently does take place; and when the patient first comes under observation as a case of heart disease, especially if the condition sometimes happens to be associated with cardiac bruits independent of valvular lesions. Of cases of temporary cardiac invalidism there must be many examples which escape detection. Now that attention is drawn to the condition it is hoped that it may bring forth valuable observations in the future on an important subject which needs further and more extended investigations.

(*Exhibited February the 19th*, 1904.)

Discussion.

Dr. Robert Hutchison said he agreed with Dr. Carpenter's view that the condition of the heart was due to diphtheritic poison, and if so it was an additional proof of the idea which was gaining ground that the cases of heart failure with diphtheria were due not, as was previously supposed, to the effect on the cardiac nerves, but to direct action on the muscle-fibre, just as people were beginning to realise that the rheumatic poison affected the heart muscle as much as it affected the valves.

Dr. C. O. Hawthorne was disposed to agree with Dr. Carpenter. He recorded some years ago a case of decided irregularity of the heart in a child with albuminuria and loss of knee-jerks, but without any history of diphtheria. And on the basis of that experience and other similar ones he would be

inclined to accept Dr. Carpenter's suggestion. Cases of sudden syncope after diphtheria were, of course, well known. Dr. Carpenter's case in all probability afforded a demonstration of the manner in which these disasters were produced.

Dr. C. W. Chapman said such cases sometimes went rapidly to the bad. He saw a child who had apparently recovered from diphtheria who had rapid cardiac dilatation with syncope. The patient rallied, but died suddenly three days afterwards.

Dr. Parkes Weber thought that in supposing the trouble to be post-diphtheritic, Dr. Carpenter had hit upon the only explanation which would satisfactorily account for the cardiac condition.

The Chairman (Mr. R. Clement Lucas) thought the case showed that the condition might easily be overlooked. He remembered a case of diphtheria dying at Guy's Hospital, and it was found there were many patients in the ward at that time suffering from slight sore throat. On scraping their throats and examining the scrapings the Klebs-Loeffler bacillus was found in almost every case, though they had no serious symptoms.

46. A FATAL CASE OF ŒDEMA ASSOCIATED WITH ALBUMINURIA IN AN INFANT AGED 8 MONTHS.

By GEORGE CARPENTER, M.D.

Maud S—, aged 8 months, was brought to the North-Eastern Hospital for Children on December the 7th, 1903, with a history of having wasted since measles, ten weeks previous. Her abdomen was prominent, and her liver extended three fingers' breadth below the costal margin in the nipple line. She was neither rickety nor anæmic, and there were no physical signs. Her mother had borne six children; she had one born dead and one still-birth, and had produced three children, living, since then. On December the 14th she was considered to be progressing well, but a week later her face began to swell, and then her hands and feet. The illness commenced with sickness every time she took the breast. She did not pass much

water, and it was said to be a "kind of brown colour." On January the 4th her face was waxy-looking; both eyes were œdematous, the right eye especially. The feet and legs were very dropsical, deeply pitting on pressure, also the trunk; and the feet were cold and blue-looking. The buttocks and groins were raw-looking and weeping. On the right cheek there was a tiny cutaneous gumma with a reddened surface, the size of buck-shot. The mucous membranes were rather pale. There were also a number of superficial lesions on the cheeks, the size of a hemp-seed, with a certain amount of surface exudation, which the mother said came out like yellow blisters. She was admitted into the hospital. On January the 7th the œdema of the trunk had increased slightly, and that of the eyes had decreased. The spots on the face were more numerous. The smallest quantity of urine which had been passed was 4 ounces in the twenty-four hours, the largest 5 ounces. The highest pulse-rate per minute was 116, the lowest 92; the highest respiration-rate 36, the lowest 24. The lowest temperature was about 97° F., the highest 97·8° F. There was a very decided cloud of albumin in the urine, and a sediment on boiling. On January the 8th the œdema was less on the dorsa of the hands, and on the right side of the trunk. Œdema was well marked on the inner side of each thigh, the fronts of the legs, and the backs of the feet. There was slight œdema round the umbilicus. The most marked swelling was on the buttocks, where several drops of serum could be seen exuding from the surface, which was red and shiny. There was no lumbar œdema. On January the 9th the urine was thick and cloudy, and contained 1 gram of dried albumin per 1000 c.c. The urea amounted to ·4375 grains per ounce. The œdema was less in the morning than at night. On January the 10th there was a slight trace of albumin, and the urea was ·4375 grains per ounce. On January the 11th there was a slight trace of albumin. The swelling was slightly less than before. On January the 12th there was a slight trace of albumin; there were no tube casts. The largest

quantity of urine that had been passed since the previous record was 5½ ounces, the smallest quantity 3 ounces. The child was in a very precarious condition; no radial pulse could be felt, and the breathing was shallow and irregular, and she died on January the 13th. The post-mortem examination was made on the same day. The brain, which weighed 23 ozs., was normal. The heart was healthy. The right lung weighed 2¾ths ozs.; it was in part œdematous. The left weighed 2½ ozs., and was in a similar condition. The liver was pale; it weighed 11½ ozs. The right kidney weighed 1 oz., the left 1½ oz.; neither appeared abnormal. The spleen weighed ¾ths oz. The stomach and intestines were normal. Microscopically nothing abnormal was detected in the kidneys. The liver was fatty; the heart was normal. Some small patches of broncho-pneumonia were found in the lungs.

Remarks.—In the absence of definite pathological changes in the organs one is forced to fall back upon the explanation of toxæmia to account for the symptoms during life, though how and where the toxin was manufactured which produced the fatal result the autopsy does not show. (*Exhibited February the 19th*, 1904.)

Discussion.

Dr. Parkes Weber thought one must acknowledge that there was nothing to indicate nephritis in the microscopic appearance of the kidney, but on the other hand, it was not the appearance met with in sections of healthy kidneys from persons killed by accidents, etc.; it was like the appearance often met with in the kidneys of persons who had died from some cardiac or other disease not associated with actual nephritis. He thought the case was an example of what could be called *toxæmic œdema* and *toxæmic albuminuria*. Absence of nephritis in such cases might be due to personal peculiarity. There was certainly a relationship between toxæmic albuminuria and nephritis. Doubtless, the same causes which in some individuals led to toxæmic albuminuria and toxæmic œdema might in other individuals set up actual nephritis. Possibly, in some cases of acute toxæmic albuminuria without nephritis evidence of actual nephritis would have been found had the patients lived longer.

47. A FATAL CASE OF STAPHYLOCOCCUS MOUTH INFECTION (ULCERATIVE STOMATITIS) FOLLOWING MEASLES IN AN INFANT.

By DAN McKENZIE, M.D.

The course of events in the following case affords a striking instance of the oft-noted lack of resistance to the invasion of certain organisms induced by an attack of measles.

K. H—, a healthy, well-nourished girl aged 18 months, was attacked by measles on March the 2nd, 1903. The illness ran a moderate course, free from complication.

On March the 29th I was sent for on account of pyrexia reaching 104° F. with a rapid pulse. There was a general blush of the skin resembling somewhat the rash of scarlet fever, but not definitely punctate in appearance. There was no sore throat. A general moderate enlargement of all the accessible lymphatic glands was observed. The mother had noticed some blood in the sputum, which she had supposed to come from the chest. No consolidation of the lungs was found, and there was no dislocation of the pulse-respiration ratio. The temperature and rash persisted for three or four days, the rash gradually fading, and the temperature falling to normal rather rapidly at the end of that period. During the next eight days the temperature was subfebrile (from 97° to 99·8° F.) Then it gradually rose to 102° F. By this time (April the 18th) a fine branny desquamation was visible. The lymphatic glands had gone back to the normal size, with the exception of two submaxillary glands on the left side, which were still large and hard to the feel. On the alveolar margin of the gum above the left lateral incisor of the upper jaw a small round ulcer was discovered, about three millimetres in diameter, with a grey rough floor and undermined edges, surrounded by an areola of red

and swollen mucous membrane. During the next few days the temperature remained high, the alveolar ulcer slightly increased in size, and the submaxillary lymphatic glands became larger and semi-fluctuant, while the peri-glandular tissues were hard and infiltrated. On April the 24th, under chloroform, the glandular abscess under the jaw was opened and scraped, about a tablespoonful of thick pus being evacuated. The ulcer on the gum was scraped with a sharp spoon, and the broken down tissue, which considerably exceeded the limits of the ulcer, reaching down to the periosteum, carefully removed. The resulting raw surface was painted with carbolic acid.

Next day there was a recurrence of the roseolous rash which marked the beginning of the illness, together with a rise of temperature to over 104° F. The case was further complicated by the occurrence of diarrhœa, which continued henceforth throughout the whole course of the illness. The events of the following month showed that little or no good had been effected by the surgical measures adopted. The submaxillary abscess cavity certainly slowly filled up, but the ulcer in the mouth gradually increased in extent and depth, while no reduction in the height of the temperature occurred. Strangely enough the child's general condition at this period underwent marked improvement, probably owing to the excellent nursing and to the ability of the patient to take and digest large quantities of nourishment.

On April the 22nd the alveolar ulcer was again scraped and painted with carbolic acid. It was found to extend from the alveolar margin of the gum above the first and second incisor and canine teeth to the junction of the mucous membranes of the gum and cheek, a very considerable increase in extent.

On May the 2nd, ten days after the appearance of the second roseolous rash, desquamation was observed.

On May the 7th the ulcer was scraped for the third time. By this time, although the abscess cavity under the jaw had filled up and the wound seemed about to

cicatrise over, there had been no absorption of the dense infiltration surrounding the enlarged glands, and the whole of the submaxillary region on the left side was occupied by a hard brawny mass, which extended across the middle line beneath the chin. There was also some enlargement of the submaxillary lymphatics on this side. The left cheek was red and œdematous, and the breath foul. A day or two later the wound became infected.

On May the 16th the red rash appeared for the third time, and was followed in due course by desquamation. During the next six weeks the local conditions underwent a very slow but marked improvement. The ulcer in the mouth ceased to spread, the cheek grew less inflamed, and the submaxillary induration showed signs of being absorbed. But the wound enlarged by a process of disintegration of the subcutaneous connective tissue.

On June the 17th the final stage was reached when the patient gave evidence of intra-cranial mischief in the form of sickness and vomiting, with transient lapses of consciousness, photophobia, retraction of the head, and stiffening of the erector spinæ muscles, while emaciation progressed rapidly. In a few days a passing paresis of the right arm and leg appeared (June the 23rd). Then clonic convulsions lasting twenty-four hours occurred, followed by coma, gradually deepening into death on June the 28th, three months after the onset of the illness.

A post-mortem inspection of the brain only was permitted. The cerebral symptoms were found to have been caused by sinus thrombosis.

A culture taken post mortem from the alveolar ulcer showed the *Staphylococcus pyogenes aureus*.

The treatment of the case consisted in the exhibition of iron, arsenic, cinchona, and remedies to meet the various complications as they arose. Potassium chlorate was tried at two different times, once in three-grain doses four times a day for six weeks, but no good effect followed its administration.

Antistreptococcus serum was injected, 10 c.cm. at a time, six times in all between May the 19th and June the 15th, generally without any distinct result, although once or twice a severe reaction with urticaria and restlessness occurred.

The roseolous rashes were presumably due to systemic infection from the infected localities. The heart and lungs remained unaffected throughout.

Mr. Jonathan Hutchinson, jr., reported some years ago a somewhat similar case to this one, in which, however, death seemed to have been directly induced by miliary tuberculosis. In his case, a child of twenty-one months, the ulcer or ulcers in the mouth persisted for nine months.*

(*Read February the 19th*, 1904.)

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48. THE TREATMENT OF HERNIA IN YOUNG CHILDREN.

By P. LOCKHART MUMMERY, B.C.

I THINK this subject is a very important one, and a profitable one for discussion in this Society.

This Society now holds an important position as an authority upon the treatment of children's diseases, and any statement of opinions which will aid in establishing a satisfactory method of treatment, especially for such a common condition as that of hernia, cannot fail to be of value.

And it is rather with the object of starting a discussion, and obtaining a definite opinion upon this important subject than with the object of bringing before you anything new or original, that I am reading this paper.

The number of cases that one meets with of hernia in

* 'Path. Soc. Trans.,' Lond., 1887, vol. xxxviii, p. 127.

infants and young children is very considerable, and it has always seemed to me that the treatment of these cases in young children by the use of trusses is very unsatisfactory.

There are several reasons for this. In the first place, it is not an easy matter with a fat baby to obtain a truss which will remain in position and keep back a hernia without having one with a very strong spring, and this is apt to result in a short time in considerable soreness of the skin, which in infants is very ill adapted to withstand pressure.

The old wool or worsted truss, which is so much vaunted in all the text-books, and which is still used as a pitfall for the examination student, is, so far as my experience goes, absolutely useless. Its only value seems to consist in the fact that it enables the child to be taken home before the hernia comes down again. Even if the wool truss were capable of keeping up the hernia, it is quite useless as a curative agent, as it does not cause sufficient pressure upon the neck of the sac to have any chance of obliterating it.

The only cases in which the use of a truss can be expected to be of any real service in curing a hernia in a child are those cases where the parents are sufficiently well off to be able to obtain the services of a skilled nurse to look after the child, and to pay for the necessary trusses. Then, if the nurse is able to change the truss without allowing the hernia to come down, and is able to prevent the child's skin from getting chafed by the constant pressure of the truss, a satisfactory result may be sometimes obtained.

However, it will be necessary for the child to wear the truss constantly, night and day, and even then the time before a cure of the hernia can be expected to take place is considerable. It is seldom, even under the most favourable conditions, that anything approaching a real cure of the hernia can be secured in less than a year, and in most cases it is two years or more before the truss can be

dispensed with, while in many cases the use of the truss fails altogether to cure the condition. So that even under the most favourable circumstances the use of a truss for the purpose of curing a hernia in a child, means that we must condemn the child to wear a truss constantly for a period of probably two years, and that even then we may quite fail to cure the hernia. And if we are successful in effecting a cure of the hernia in the sense that it no longer comes down, we leave the child with a condition which in many cases predisposes to hernia later on in life. For the treatment with a truss, even if successful in curing the hernia, still leaves the sac partly unobliterated, since in most cases the sac is congenital and cannot be returned into the abdomen. This leaves the child with a hernial sac which is ready at any time to receive a hernia should the abdominal wall give way. And, in fact, the child is predisposed to the recurrence of the hernia later on in life.

That this may occur is shown by the following case :

A sailor boy, aged 14, came to St. George's recently to be operated upon for a scrotal hernia which had appeared after a strain two months previously. His history showed that he had had an exactly similar hernia when an infant, and that it had been apparently cured after wearing a truss. I suppose the neck of the sac had been partly obliterated by the pressure of the sac, sufficient to keep up the hernia, but when the boy began to lead an active life the adhesions in the neck of the sac were no longer able to bear the strain, and the old hernia recurred.

And although it is very difficult to obtain any statistics upon this point, the conditions found in a large number of cases of hernia in adults seem to show that this occurs much more commonly than is generally supposed.

As regards the use of a truss for the treatment of cases met with in hospital practice, I think that it is very rarely of any use. The mother is seldom sufficiently intelligent to be able to change the truss without allowing the hernia to come down, and even if she is, the expense

of two trusses to start with and the subsequent new trusses and repairs that will be necessary renders it impossible in most cases to satisfactorily carry out the treatment. As a rule one finds that the hernia constantly comes down behind the truss, and that the latter is simply pressing upon the neck of the sac; in which case the truss is not only useless but is a source of danger, as it predisposes to strangulation.

When, as is the case with children and infants, the object of the truss is to cure the hernia by causing obliteration of the neck of the sac, it is most important that the hernia should not come down at all. And this requires much more care and attention than most out-patient mothers are able to afford.

I think the most satisfactory method of treatment of these cases is by operation.

The objection that surgeons had against operating upon young children is almost a thing of the past, and we now know that children stand operations almost as well as adults, providing always that certain precautions are taken.

The treatment of hernia by operation has two great advantages.

First, that it gets rid of the sac, and, secondly, that it enables us to cure the hernia in the space of about three weeks, and does not necessitate the wearing of an instrument by the child.

The operation which I have performed in these cases, and which I am inclined to think is a very suitable one, is a modification of Barker's operation. The usual incision is made, and the sac separated up to the internal ring. The sac is opened, and with one finger inside to act as a guide and to keep back the intestine, the neck of the sac is ligatured over the point of the finger as high up and as near to the internal ring as possible. The sac is then cut away on the distal side of the ligature. The peritoneum is separated slightly with the point of the finger from around the internal ring. One end of the ligature is threaded upon a hernia needle of suitable

size, and the needle is passed into the internal ring and made to perforate all the thickness of the abdominal wall from the inside, so as to emerge about half an inch above the ring. The other end of the ligature is threaded and brought through the abdominal wall in a similar manner, so as to emerge a little distance from the first end of the ligature. Then, by drawing upon the ends of the ligature, the neck of the sac is drawn away from the internal ring, and the ends are tied together, so as to retain the neck of the sac in its new position.

The internal ring is not stitched up. I think that, at any rate in infants, it is neither necessary nor advisable to stitch up the ring; the tissues are very delicate and badly adapted for stitching, and if the cause of the hernia, namely, the congenital sac, is removed, the ring itself soon contracts and gets rid of any weak spot in the abdominal wall. Moreover, by not stitching up the ring a good deal of time is saved, and this is a matter of great importance when operating upon young children. In older children, or where the ring is very large, it is better to close the ring with one or two stitches. In many cases the operation may be simplified by not dissecting out the scrotal portion of the sac, but dividing it above.

This operation is very quickly performed, and this is a desideratum of the greatest importance in operating upon infants. The chief difficulty in the operation is in defining the sac, which is often extremely thin and delicate.

In all these cases the after-treatment of the case is a most important factor, and the chief point is, I think, the position in which the child is nursed.

The best position in which to nurse the child after a radical cure of hernia is with the legs slung up to a cross-bar.

Stirrups are attached to each leg by means of extension strapping, and both legs are slung up to a horizontal bar over the top of the cot, in much the same way as in treating a fracture of the shaft of the femur by Bryant's method.

This position has several advantages:

1. It is comfortable, and at the same time prevents the child from moving about to an undesirable extent.

2. It enables the child to be kept clean easily; a matter of no little importance in these cases.

3. It affords very valuable support to the inguinal rings, and prevents an undue amount of pressure being thrown upon them just after the operation by the child crying, etc.

4. It relaxes all the tissues in the neighbourhood of the wound and so allows healing to take place rapidly and effectually.

As regards the age at which the operation can be performed I do not think that one can fix any limit.

The youngest child I have operated upon for hernia was six months old, though I know that other surgeons have operated upon younger children.

I think, however, that it is seldom necessary, or even advisable, to operate upon very young infants for this condition. Apart from other considerations, the operation is rendered much more difficult owing to the smallness of the parts and the extreme delicacy of the sac, which is often very difficult to find.

The hernia can be safely left to itself, and so long as no truss is applied there is very little risk of strangulation. Later on, when the child is a year or eighteen months old, the operation can be performed and the hernia cured.

The risks of the operation are very slight. I do not myself know of a single case which ended fatally, or in which there was any serious complication.

I have collected thirty-nine cases of radical cure of hernia in infants and young children, and among these there was no case that ended fatally and very few cases in which there were any complications. Out of the whole thirty-nine cases, swelling and inflammation of the testicle, on the side on which the operation was done, occurred in one case, and the swelling very quickly cleared up.

The formation of a hæmatoma in the wound occurred

in two cases, and in one case the wound suppurated and there was a septic rash for a few days, which gave rise to a suspicion of scarlet fever, but it cleared up in a few days without any untoward symptoms. In several of the cases treatment by a truss had been tried and had failed.

Since I commenced to write this paper, one upon the same subject has been published in the 'Lancet' by Mr. Robert Campbell, and in it he reports 114 cases of radical cure of hernia in young children without a single death, and with very few complications. So that the risks of the operation for radical cure in infants and young children do not seem at any rate to be greater than when the operation is performed upon adults, that is to say, very slight indeed, and certainly less than the risk which a child runs from strangulation when wearing a badly fitting truss. (*Read February the 19th*, 1904.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said he presumed Mr. Mummery did not suggest that the operation he detailed was an original one, because it was the operation he had himself performed for quite fifteen years. With regard to performing the operation on young children, there was one special difficulty, namely, that of isolating the vas deferens from the sac. It was so tiny and narrow, and adhered so closely to the sac, that there was a real danger of its destruction in inexperienced hands.

Mr. Lockhart Mummery, in reply, said it was precisely that difficulty referred to by the Chairman which he alluded to, and which was a very real one; also, the sac was so extraordinarily thin that it was difficult to see it. He of course did not intend the operation he had described to be considered an original one, but simply indicated that it was the operation which he regarded as the best.

49. A CASE OF HEMI-HYPERTROPHY IN WHICH THE INTERNAL ORGANS WERE AFFECTED.

By ROBERT HUTCHISON, M.D.

Thomas F—, aged 4 months, was the fourth child of the family, the three others being healthy. He was born at full-time after an easy labour, and his mother's health during her pregnancy was good. The condition about to be described was noticed at birth.

He was a healthy, well-developed child. There were three small capillary nævi in the skin, one above the right knee, one in the left lower axilla, and the other below the left scapula, but with that exception he presented no abnormality save for the condition of the left arm and leg. The viscera were healthy. The asymmetry involved the left arm and leg, and to some extent the trunk also, but the head, face, and tongue were quite symmetrical. There was no abnormality of the digits. The asymmetry of the limbs was apparently due to an increase in the subcutaneous tissues of the left side; the condition in the leg, particularly, closely resembling a diffuse lipoma. Measurements showed that the left forearm was an inch and a half greater in circumference than the right, the left calf two inches greater than the right, the left thigh an inch and a half larger than the right. There was no difference in the length of the limbs on the two sides. The left chest had a circumference of one inch more than the right at the level of the nipples, whilst the left side of the abdomen at the level of the umbilicus exceeded the right by an inch and a quarter. The skin on the left side showed no difference in colour from that on the right.

The case was regarded as one of hemi-hypertrophy of the "false" sort, *i. e.* involving the soft parts only, and not affecting the bones. Shortly after he was first seen

the child was attacked by broncho-pneumonia and left-sided empyema, to which he succumbed. A careful post-mortem examination was made by Dr. Salaman, of which the following are the chief results :—

The increased thickness of the left limbs and left side

Fig. 13.

of the trunk was due entirely to an increased deposit of subcutaneous fat. There was no evidence of any nævoid tissue or vascular dilatation. The bones on the left side were no thicker than on the right. The brain was normal and symmetrical, and the pineal and pituitary bodies presented no abnormality. On the other hand, most of the paired organs were decidedly larger on the left side

than on the right. This is shown by the following weights:—Left kidney, 56 grammes, right, 28 grammes; left suprarenal, 42 grammes, right, 14 grammes; left testicle, 2·3 grammes, right, 0·55 grammes. The lungs could not be satisfactorily compared owing to the presence of a left-sided empyema and pneumonia. The heart seemed normal in its development, and the two lobes of the thyroid were symmetrical. On the other hand the left lobe of the thymus was decidedly larger than the right, but their exact relative weights do not appear in the report. The liver presented the usual degree of relative development of its left and right lobes, but contained some multiple angiomata.

Remarks.—Post-mortem examinations on cases of hemi-hypertrophy are so rare that the above case seemed worth recording. The asymmetry of the internal organs was most striking, and suggests that the hemi-hypertrophy in such cases cannot be the result of any mere "trophic" influence, but must date back to embryonic life, and be the consequence of unequal segmentation in the ovum.

(*Exhibited February the 19th*, 1904.)

Discussion.

The Chairman (Mr. R. Clement Lucas) said the Society was greatly indebted to Dr. Hutchison for showing the remarkable specimen. He, Mr. Lucas, had been in the habit lately of operating upon solid œdema, where it had resulted from venous or lymphatic obstruction existing for a long time, by excising large pieces of cellular tissue. In that way the hypertrophied fatty condition shown in Dr. Hutchison's case was capable of cure.

Dr. C. O. Hawthorne congratulated Dr. Hutchison upon the specimen; it was extremely rarely that one could verify the one-sided enlargement of the internal organs in these cases. He knew of an instance in which all the external parts were larger on one side than the other, this extending even to the tonsil, and Dr. Hutchison's demonstration showed that a parallel condition was to be found in the viscera. He would be interested to hear whether the blood-vessels were unduly large on the hypertrophied side, because that would have some bearing on the question of causation. Around that subject there had been some amount

of controversy. Some held that enlargement of blood-vessels and corresponding increase of blood supply was the primary fact, the hypertrophy of other tissues being thus a result of "over-nutrition," while others held that the enlargement of the blood-vessels was simply part of a general hypertrophic process. In the case of a boy he had in mind and recorded by Dr. A. N. Macgregor, of Glasgow, the hypertrophy was limited to the lower limb, and chiefly below the knee. The request was made that the limb should be amputated, and that was done, and afterwards the boy fell into a stuporose condition and died. Post-mortem there was discovered a tumour in the brain, involving the optic thalamus. In view of the relation between the enlargement of the pituitary body and the condition of acromegaly this case of Dr. Macgregor's seems of much importance.

Dr. F. Parkes Weber said the case furnished an excellent example of the association of congenital (or rather developmental) general overgrowth of a portion of the body with angiomata in the skin representing vascular overgrowth. In the present case moreover there were angiomata in the viscera (liver) as well as in the skin. Dr. Hutchison had told him that the nævus-formation in the liver was characterised by the presence of abundant endothelial-like cells. He mentioned that because the hepatic angiomata in Dr. Hutchison's case were evidently more allied in structure to the growing nævus of the skin in children than to the ordinary nævus of the liver in adults.

Dr. Robert Hutchison, in reply, said there was no obvious vascular dilatation in the subcutaneous tissues. There was not very much attention paid to the size of the deeper vessels. It seemed almost impossible to say whether the vessels were big because the organs were big, or whether the large vessels had led to the hypertrophy of the organs. The nerves were not examined. He showed a giant finger which had been amputated, and also an enlarged supra-renal capsule on the same side from the same patient.

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50. A CASE OF ENLARGED LIVER AND SPLEEN.

By HAROLD BURROWS, M.B.

The patient was a girl, aged 13 years. Six months previously a laparotomy had been performed for supposed hydatid disease of the liver. At the operation the

liver was found to be uniformly enlarged, and to have a perfectly smooth surface. No family history could be obtained, and nothing could be learned of the patient's past history except what she herself could give, and she had been unaware of the swelling until brought to hospital by a neighbour for pain in the abdomen.

The child now appeared to be in good general health. She was small for her age, the skin of the face and forearms was dry and scaly, and there were remains of old retino-choroiditis in both eyes. A blood-count showed 5,500,000 red corpuscles and 36,000 white corpuscles per c.mm. Otherwise there was no abnormality to note beyond the marked hepatic and splenic enlargements. The cause was probably congenital syphilis. It would be interesting to know what was the likelihood of serious symptoms ultimately developing.

(*Exhibited March the 18th*, 1904.)

Discussion.

Dr. Leonard Guthrie agreed that the case was probably syphilitic, and he thought there might be gummata in the liver and spleen.

Dr. Porter Parkinson said the case reminded him of one shown at the Clinical Society by Sir Thomas Barlow in an adult. But that patient had had an enlarged liver and spleen ever since birth; it was discovered almost immediately after birth, and there were definite symptoms, because the patient suffered from recurring attacks of pain in the abdominal region, also from jaundice. There was a history in the mother and in some other relatives of a similar condition of enlargement of the liver and spleen. In one case of a similar nature exploration of the abdomen was carried out, but nothing except the enlargement could be discovered. No treatment seemed to be of any avail. The feel of the liver and spleen rather suggested a uniform enlargement than the presence of a tumour or gumma. The liver substance appeared to be of about the normal softness and its surface smooth.

Mr. Harold Burrows, in reply, said that at the operation the surface of the liver was found to be smooth, and there were no healed gummata.

51. RECURRENT CARDIAC DILATATION TREATED BY ADRENALIN SOLUTION.

By C. W. CHAPMAN, M.D.

J. P—, aged 12 years, came under treatment as an out-patient in June, 1903, for mitral disease consequent upon rheumatic fever. After doing well, she in the following September became worse, and was admitted into hospital. The heart was found dilated and the action feeble. She promptly responded to treatment, so that she was able to leave the hospital in a month. The patient continued in fair health until the early part of this year. The symptoms becoming severe, she was readmitted on January the 19th. The heart was dilated to fully two fingers' breadth to the right of the sternum and 1½ fingers beyond the left nipple line. The liver was down to the umbilicus, and the dyspnœa was very urgent. Treatment on the usual lines failing to be of much benefit, adrenalin was prescribed in 5-drop doses every four hours. An improvement in the symptoms—notably the breathing—occurred after a few doses, and the patient has steadily gained ground.

(*Exhibited March the 18th*, 1904.)

Discussion.

Dr. Fortescue-Brickdale (Bristol) said it would be interesting to know how long the child had been on adrenal extract, and whether there was any other treatment carried out at the same time, *e. g.* keeping the patient in bed, or using massage or exercises.

Dr. C. W. Chapman replied that no other treatment was carried out in the case. The child had been kept in bed a fortnight before the extract was tried. There was not even any alteration of diet during its administration. The chief symptom was orthopnœa, in consequence of which the child had to be propped up in bed.

52 AUTOMATISM WITH OLFACTORY AND GUSTATORY AURÆ.

By LEONARD GUTHRIE, M.D.

The patient shown was a boy aged 11 years. For 2¼ years he had been subject to peculiar attacks in which he would suddenly run to his mother, or if in school to his teacher, exclaiming, "Horrid smell!" or "It's the smell!" He would appear much agitated and distressed, though not terrified, for a few moments, sometimes spitting and grimacing, and going through the action of trying to pull something out of his mouth. He would then behave "as if he were silly," running to persons in the room and trying to kiss or embrace them, and talking nonsense meanwhile. If restrained he would cry "Don't touch me!" and struggle. This would go on for five to ten minutes, after which he would become quiet, turn pale, shiver as if cold, seem exhausted, and lie down to sleep. On waking subsequently, he would invariably complain of severe headache. He never seemed unconscious at the time of an attack, but afterwards he remembered nothing that he had said or done. He only knew that he had experienced the "horrid smell," which he could not otherwise describe. Similar attacks occurred once or twice weekly for two years, but he had remained free from them for so long as three months at a time. He never wetted the bed, but sometimes at night he would jump out of bed and go through the same performance as by day. Occasionally, instead of the evil smell, he would complain that "something was running about in the back of his head," and his father had instructed him to shake his head violently when he felt the sensation coming on, in order to arrest further progress of the seizure. This method was sometimes successful. He also had seizures resembling petit mal at times, but had never been

convulsed since infancy, when he had as many as seven fits a day whilst teething.

At the time when the peculiar attacks first made their appearance he had had tonsils and adenoids removed by operation. His appetite and digestion were good, and he had had no serious illnesses, but frequently complained of headache.

The boy was nervous-looking, slightly built, with rickety forehead and bright prominent eyes. Optic discs were normal, teeth ridged and deficient in enamel; heart and lungs healthy. He was very intelligent and did well at school. There was no family history of rheumatism or any nervous diseases.

Under bromide of ammonium, in 8-grain doses three times daily, the fits decreased rapidly in frequency and severity.

In January, 1903, he had two slight attacks, and another in March; a bad one occurred in May, after which the dose of bromide was increased to ten grains. He then remained free till October, when after discontinuing the medicine for a month, two slight seizures were reported. He had now been free from them for nearly six months.

Olfactory auræ are rare in epilepsy, and gustatory auræ still more so. Sometimes, when due to organic disease, this had involved the uncinate gyrus. There was no evidence of gross cerebral mischief in this case, though no doubt there was functional disturbance of that particular area of the brain. The seizures themselves resemble "ambulatory automatism" rather than true epilepsy.

This suggested comparison with "day terrors," which usually occur at an earlier age than this. In these, a child of five or six would suddenly stop in the midst of play and rush to its mother with an appearance of liveliest terror, shrieking, "Something black!" or "Someone is after me!" or stop its ears and cry "I'm afraid!" There was, as a rule, no loss of consciousness, but only uncontrollable fear. An attack lasted 5–10 minutes to

half an hour. It ended in exhaustion and sleep, usually followed by intense headache.

The attacks were precisely similar to "night terrors," except that they occurred during waking hours. In some cases he believed the hallucinations were due to the auræ of migraine occurring in highly emotional and timid children. (*Exhibited March the 18th*, 1904.)

Discussion.

The Chairman (**Dr. Percy Lewis**) (Folkestone) said he noticed the pupils were very large, and he thought there was a tendency to exophthalmos.

Dr. Porter Parkinson asked whether any results from treatment had been derived in the case, by bromides or other drugs.

Dr. A. E. Jones said the gustatory auræ seemed to be unknown to the mother. He asked whether they were pleasant or unpleasant. Visual auræ could be divided into two kinds—(1) the auræ of persons, which were usually unpleasant, and (2) the auræ of landscapes, which were generally pleasant. The auditory auræ were usually pleasant, but olfactory unpleasant.

Dr. Leonard Guthrie said the discs were normal, but he would not answer for any error of refraction which might be present. The pupils were generally much dilated when the boy was excited. The treatment in the case had been satisfactory. When the boy first came to him he had been subject to the attacks once or twice weekly for two years, but under treatment they rapidly subsided, both in frequency and severity. He gave 8-grain doses of bromide of ammonium. That was left off and he had a bad seizure. He then resumed it and increased it to 10 grains, and he had had no further seizures for six months. He thought the gustatory auræ must have been unpleasant because the boy made grimaces and spat about, as if he had something nauseous in his mouth.

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53. MULTIPLE CONGENITAL DEFORMITIES.

By EDMUND CAUTLEY, M.D.

The patient, a girl aged 5 years, is the eleventh of twelve children. Of these three are dead. There is no

history of congenital defects in any other member of the family, and no history of syphilis. The mother has had two miscarriages. The child was born at full-time and breast-fed up to the age of two years.

She is very small and intellectually deficient. Her speech is limited to a few words, and her comprehension is rather deficient. She cannot walk or stand, although the lower limbs present no abnormality sufficient to account for the disability. There is some genu valgum and the limbs are rather small, but there is no rigidity; the electrical reactions are normal and the reflexes are unaltered. Her mode of progression is by alternating movements of the buttocks, while sitting on the ground.

The right eye shows slight notching of the upper lid at the junction of the inner and middle thirds, and two small dermoids in the outer and lower segments of the conjunctiva.

The left eye exhibits a more marked notch, in the same situation as that on the right side; a large coloboma of the iris, involving the choroid; a red optic disc and a patch of white choroidal atrophy. The right angle of the mouth is imperfect, the lips not being properly united, and the fissure prolonged into a groove on the inner side of the cheek.

The ears are well formed, but on each side, anterior to and separate from the ear, is a well-marked accessory auricle, larger on the left side. The one on the left side contains cartilage, and between it and the ear are two smaller tubercles.

No cervical fistulæ or cysts are present.

In the sacro-coccygeal region there is a very distinct sacro-coccygeal dimple.

Generally the child shows evidence of moderate rickets. The frontal eminences are prominent, the calvarium somewhat square, and the fontanelle barely closed. The teeth are much decayed, the lower radial epiphyses are enlarged, and the ribs show some beading.

The case is of interest by reason of the multiple and

varied defects and the absence of anything in the family history which can be elicited to account for the condition. It is very curious that only one child in such a large family should present defects. Although one of the latest born, she is not the youngest, and the latter is perfectly formed.

(*Exhibited March the 18th*, 1904.)

Discussion.

Mr. Lockhart Mummery said a most interesting point was the association of auricular appendages with the fissure of the mouth, because such cases seemed to bear out the theory that the auricular appendages were developed around the outer end of a partly persistent inter-maxillary cleft, the anterior portion of which was represented by the fissure of the mouth. Two cases were reported by Mr. Morgan before the Royal Medical and Chirurgical Society some years ago, in one of which the child had a cleft of the mouth similar to Dr. Cautley's case, but it extended back to the anterior border of the masseter, and was associated at the posterior end with an appendage similar to the one in the present case; there was also an auricular fistula which ran downwards and forwards for $\frac{1}{4}$ths inch. The anterior part of the inter-maxillary fissure had failed to join, and resulted in the fissure of the mouth, while the posterior portion had developed into the auricular fistula. If this theory was correct, one ought to find cases in which the entire inter-maxillary cleft had failed to close, and in the College of Surgeons Museum there was a sheep's head in which that failure was seen. The first arch was missing, and with it the cleft was completely patent; the mouth was completely open into the ear on that side, but the ear was normally developed. The theory of such conditions was that the first branchial arch secondarily divided anteriorly, the lower portion forming Meckel's cartilage and the upper one the upper jaw; and between these two secondary arches in the embryo there was a secondary fissure which gave rise to the conditions under discussion. The theory which had been put forward, that the supernumerary auricles were due to aberrant tubercles of the ear which had developed wrongly, could not be held. If it were true, the ear itself should be abnormally developed; but in the present case the ear was perfect, except that it had the extra condition of appendages in front.

Mr. Howell Evans said that apparently whenever there occurred a macrostoma there was some alteration at the posterior part of the first cleft, though the auricle itself might be normal. When the macrostoma was seen in such a child as this, one

could anticipate finding on that side or on the opposite side some pre-auricular tubercle or appendage, but where there were pre-auricular appendages it did not necessarily follow there would be a macrostoma. The theory of the formation of the upper and lower jaw mentioned by Mr. Mummery was the one usually accepted, and it was probably associated with the division of the nerves, as in the case of the intercostal nerve, into a pre-trematic and post-trematic branch. On everting the angle of the mouth in all infants one could see a small tubercle which seemed to fill up the gap between the upper and lower lips. It was present in this child, but in the lower part, which proved that the upper jaw was defective; we ought to expect such, inasmuch as the offshoot is more likely to be defective than the trunk. Another point was the small niche or notch which was seen on the superior palpebra, particularly of the left eye. It occurred at the point where there was a division between the nerve supply of the outer two thirds and the inner third, a situation which was characterised in many individuals by the presence of birth marks, moles, etc. Mr. Furnivall recently had a case with macrostoma and pre-auricular appendages under his care at the London Hospital. With regard to the hyo-mandibular cleft being often very marked, one gentleman present could tell the Society about a specimen relating to it in the museum at Bristol.

Dr. Fortescue-Brickdale (Bristol) said in the specimen referred to the child was supposed to have breathed a few times after birth. It had a persistent hyo-mandibular cleft, complete absence of the lower jaw, and two small auricles at each end of the cleft. The specimen had never been dissected.

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54. A CASE OF CHRONIC INTERSTITIAL NEPHRITIS.

By G. A. SUTHERLAND, M.D.

The boy was 8 years old. There was no family or personal history of syphilis. The illness commenced with attacks of headache, vomiting, and prostration twelve months previously. The urine was of a low specific gravity; it was normal in amount, and contained at times a trace of albumin and a few granular casts. The radial arteries, and the branchials, were markedly thick-

ened, and showed increased tension. The heart was enlarged and hypertrophied, there was marked præcordial heaving, visible and palpable, and the second sound at base and apex was markedly accentuated. There were no retinal changes and no marked thirst, but the boy was wasting steadily, and the headaches were increasing in frequency. Although there was no syphilitic history in this case, he thought the most common cause of granular kidney in children was syphilis. Probably other diseased blood conditions might induce it. The prognosis in the present case was deemed to be bad, as the affection seemed to run a rapid course in children.

(*Exhibited March the 18th*, 1904.)

Discussion.

Dr. C. W. Chapman asked whether the headache was occipital or frontal.

Mr. Sydney Stephenson asked whether Dr. Sutherland examined the retinal arteries as well as the optic disc. In a case such as that described, where there was considerable thickening of the arteries, it was quite possible that some of the retinal vessels might show that change. The matter was of some importance from both a general and an ophthalmic point of view.

Dr. Leonard Guthrie said that ten years ago such a condition would have been regarded as a rarity, but since attention had been drawn to it the cases were not so uncommon as they were supposed to be. Some years ago he discovered records of only seven or eight cases; but Dr. Sawyer, of Birmingham, when writing on the subject recently, collected records of twenty-four, thirteen of whom were under 10 years of age. Polyuria and thirst might occur later in the present case. He believed, with Dr. Sutherland, that the majority of such cases were due to syphilis, though possibly there might be some other cause at work. It might be there was some congenital deficiency, not syphilitic, which caused deterioration of the blood, and that the deterioration reacted upon the vascular system. But possibly there might be cases of interstitial nephritis in very young infants which were not syphilitic, and which might go on to the chronic variety. It was often a very difficult matter to get to the bottom of such cases. Only about 30 per cent. of adults with well-marked tertiary symptoms could be induced to confess

that they had had syphilis, and in the case of their offspring they would be far less likely to own up to the truth.

Mr. Thomson Walker agreed with the remarks of Dr. Guthrie and Dr. Sutherland about syphilis being an ætiological factor in nephritis, and as to the frequency with which it was probably a cause of interstitial nephritis. He asked whether in the present case there was any suspicion of a septic focus in any part of the body. He suspected that might be the origin of some of these cases of interstitial nephritis. He believed that interstitial nephritis frequently accompanied septic processes, and that in only a few cases it reached the stage of suppuration. Some of the remaining cases might, he thought, go on to chronic interstitial nephritis.

Dr. Fortescue-Brickdale (Bristol) said a case was described four or five years ago by Dr. Bryant and Dr. Hale White, which died when a few months old with general arterial sclerosis all over the body. There was no positive evidence of syphilis observable, and if the condition had not been so marked and the child had survived, possibly the same urinary changes might have been observed as in the present case.

Dr. G. A. Sutherland, in reply, agreed that syphilis was a causal factor in a number of such cases. He believed there would be no difference in appearance between a syphilitic and a non-syphilitic kidney, because it was a question of some blood irritant. It was not very much out of the way to assume that there might be syphilitic nephritis, because there was a well-recognised condition of hepatitis and splenitis. He could not find any septic focus. With regard to the retinal arteries, Mr. Arnold Lawson had examined the case with him, and all he could make out was that the arteries seemed thin. The headache was vertical.

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55. A SPECIMEN OF EARLY PARROT'S NODES FROM A CHILD AGED 2 YEARS.

By E. P. BAUMANN, M.D.

The specimen is of interest (1) as showing Parrot's nodes in their earliest stages, when the nodes are small, sharply circumscribed, and still vascular; (2) on account of the rarity of the appearance of Parrot's nodes at so late an age as two years.

The nodes, which vary from a pin's head to a half-crown in size, are situated on the frontal and parietal bones, well away from the centres of ossification. The child from whom the specimen was taken suffered from congenital syphilis. It presented all the typical features of this disease, including enlargement of the spleen.

(*Exhibited March the 18th*, 1904.)

Discussion.

Dr. George Carpenter said the specimen of Parrot's nodes was interesting to him in several respects, firstly in that the case was obviously syphilitic, and secondly in relation to the fact that the nodes were not situated round the anterior fontanelle in the typical hot-cross-bun style, but were irregularly disseminated in other parts of the skull. It was usual to meet with the hot-cross-bun arrangement, or perhaps frontal bosses, which in either event might be associated with small nodes scattered elsewhere about the skull. With regard to their causation, within recent years the teaching had been in this country and in America also that they were due to rickets, and not to syphilis. Parrot himself was certain that they were due to syphilis, and so described them, but after his day the view altered, and the rickety explanation was accepted. Some years ago he, Dr. Carpenter, published forty cases* of the condition, and his researches had led him to the conclusion that although perhaps the majority of the children were rickety, in only about half the number of such cases was rickets either marked or very pronounced. If one were hypercritical in examinations, what child was there in whom there could not be detected at least some evidence of rickets, if one included slight beading of the ribs? It was exceedingly difficult to find in cases of cranial osteophytes corroborative evidence of syphilis, and the older the child the less was such evidence forthcoming. He exhibited skiagrams which had been taken by Mr. Sale Barker of a child with Parrot's nodes, who apparently was also the subject of a tibial node, but this node was found to be a complete fracture which had gone through both leg bones. It was impossible to determine that by any ordinary examination. In his series of cases he found 57 per cent. of them were definitely syphilitic. Last year he showed a child to the Society who had scurvy, chronic snuffles, and craniotabes, and a discussion was provoked as to the causation and exact clinical meaning of craniotabes and chronic snuffles, and a variety of opinions were expressed. That child lost its craniotabes while under treatment, and did *not*

* 'Syphilis of Children,' by George Carpenter, M.D. See pp. 70–74.

become more rickety, but subsequently developed typical Parrot's nodes. Another point was that in his series of cases at least half of them had enlargement of the spleen, and he had in his book drawn special attention to splenomegaly in association with Parrot's nodes. He had no doubt that cases of Parrot's nodes were syphilitic, and whenever he found those nodes on a skull he felt certain that child was syphilitic.

Dr. E. P. Baumann, in reply, said he was interested to hear that Dr. Carpenter held the view that Parrot's nodes were syphilitic. The spleen in the case was distinctly enlarged.

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56. A SPECIMEN OF AN EMBRYONIC CYST OF THE MESENTERY.

By E. P. BAUMANN, M.D.

The infant when 8 months old presented the symptoms of acute intestinal obstruction, and was admitted into Great Ormond Street Hospital as a case of intussusception. On operation a cyst was found between the layers of the mesentery kinking the lower end of the jejunum. On drawing the cyst out of the wound, the intestines above the level of the tumour, which had previously been collapsed, became at once distended. The child never rallied from the operation. The cyst, which was of the size of a large pigeon's egg, contained a quantity of viscid fluid in which cholesterin crystals were present. The wall of the cyst, which was ¼th inch thick, was found histologically to be composed entirely of fibrous tissues; no epithelium or muscular tissue was present. The cyst was one which a few years ago would have been called a chylous cyst of the mesentery. Since the work of Dowd, however, all cysts of the mesentery must be considered as embryonic in character.

(*Exhibited March the 18th*, 1904.)

Discussion.

Dr. George Carpenter said with regard to the mesenteric cyst case, he also had met with such an example during life. A

small infant came under his care for ill-health, and he found on examination an abdominal tumour about the size of a Tangerine orange, which fluctuated, and was associated with some enlargement of the neighbouring lymphatic glands, which could be felt by bimanual examination. The question was whether it was a chronic tuberculous abscess with tuberculous infiltration of the glands. He had an abdominal exploration made, and the tumour was found to be a mesenteric cyst, and the child made a perfect recovery. The abdominal glands were found at the operation as described at the clinical examination, but there had been only simple hyperplasia.

Dr. E. P. Baumann, in reply, said the glands in the mesenteric cyst case were enlarged, but they showed no evidence of tubercle, and that seemed to have been the case in most of the instances recorded.

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57. A CASE OF GLIOMA OF THE PONS.

By G. A. SUTHERLAND, M.D.,

AND

EARDLEY HOLLAND

(Introduced).

Willie C—, aged 6½ years, was admitted into Paddington Green Children's Hospital, on January the 8th, 1904. He had enjoyed good health up to six weeks previously, when he received a blow from a brick on the back of his head. There was no apparent bruise or bump or any definite symptom until three days later, when he complained of headache referred to the site of injury. The headache persisted, and three weeks later the right eye was noticed to be squinting. Other symptoms followed in rapid succession, namely, squinting of the left eye, unsteadiness in walking, indistinct and slow speech, dribbling of saliva from the mouth, immobility of the right side of the face, and occasionally difficulty in swallowing. There was nothing of importance as regards the patient's previous health, or in the family history.

There was definite paralysis of both sixth and both seventh nerves. The eyes could not be tightly closed,

and the whole face was motionless and masklike. Saliva was constantly dribbling from the angles of the mouth. These points are well shown in Fig. 14. The paralysis of the right side of the face and of the right external rectus muscle was complete, while a slight degree of movement still existed on the left side, this affection having been of later development than the former. The tongue was not affected, the soft palate was paretic and anæsthetic, and the voice was nasal in character; speech was delayed and deliberate. The head dropped forward as if from weakness

FIG. 14.—The expressionless face from paralysis of the external recti and facial muscles on both sides.

of the posterior cervical muscles. The gait was ataxic and feeble; he showed a tendency to fall down, and was unable to stand with his eyes closed. The knee-jerks were exaggerated on both sides, the plantar reflexes were of the extensor type, and ankle-clonus was not elicited. The boy's facial appearance suggested mental vacuity, but although dull and languid there was no evident defect of intelligence. He did not complain of any pain, but when questioned on the subject he said his head pained him, and located the discomfort in the posterior part of the right side. Examination

of the retinæ and of the ears showed no objective signs of disease. He was able to swallow well and had no vomiting.

The condition during the remaining six weeks of his life may be summed up as follows: A week after admission he had two severe attacks of cardiac syncope, from

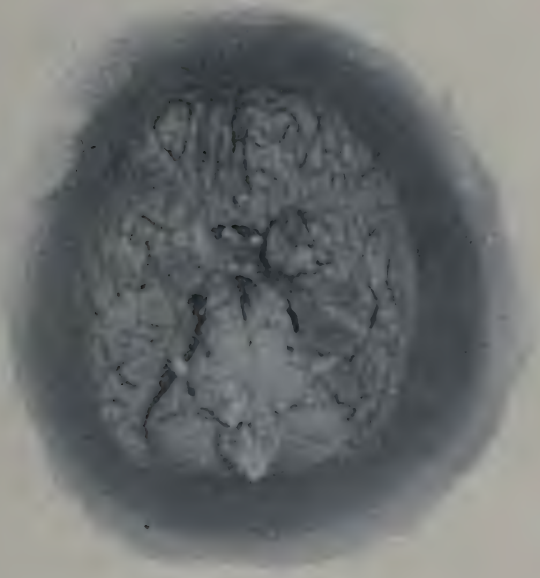

FIG. 15.—The under surface of the brain with pontine new growth.

which he rallied under stimulation. This was accompanied by great prostration, but after some days he rallied and seemed to recover more muscular power generally, and more power in the facial muscles, than he had on admission. This improvement was only temporary, and he gradually lapsed into a more torpid condition, was unable to sit up in bed, or to swallow without effort, and lost all control of the sphincters. The day before his death attacks of respiratory failure began, for the relief of which artificial respiration had to be adopted, but on February the 18th cardiac failure also supervened and he sank

rapidly. The temperature had been for the most part subnormal, and there had been no attacks of pyrexia.

At the necropsy on the day after death the convolutions of the brain were found to be markedly flattened. There was no excess of fluid in the subdural space, and the cerebral veins were not congested. The ventral aspect of the pons presented the appearance of a lobulated mass, and was considerably enlarged (Fig. 15). The colour was paler than normal and the consistency varied, being in some parts soft and jelly-like and in others harder than brain tissue. The mass extended forwards to the anterior limit of the pons and slightly on to the left crus cerebri, where it was marked by several small lobulations. The third and fourth nerves were not involved. Laterally it extended for a considerable distance on each side, the growth apparently terminating at the level of the fifth nerves, which were not involved. The cerebellum was not invaded. Posteriorly the limits varied; on the right side the tumour extended well on to the surface of the medulla, while on the left side the medulla was quite free from growth. The eighth nerves were not involved. The superficial origins of the seventh nerves were surrounded by tumour substance, whilst the sixth nerves were completely embedded. The right sixth nerve could not be found, and on the left side the nerve was seen in a groove between two lobules of the tumour. The origins of the ninth, tenth, eleventh, and twelfth nerves were not implicated on either side. The basilar artery was completely surrounded by the tumour. The lateral and third ventricles were greatly distended with fluid. The floor of the fourth ventricle was markedly flattened, due evidently to the pressure of the tumour upwards against the middle lobe of the cerebellum. The growth was an infiltrating one. No capsule and no adhesions were present. On naked eye section the limits of infiltration could not be determined, owing to the similarity in colour between the tumour and the normal tissue of the pons. On microscopic section the tumour was seen to be a glioma, which had

infiltrated the substance of the pons, the right side being more invaded than the left. Some of the pyramidal fibres were completely surrounded by infiltrations of the growth.

There was a history of injury in this case, as will usually be elicited in cases of cerebral lesions, but probably the connection between it and the development of symptoms was purely accidental. The symptoms pointed clearly to a pontine lesion, and the only question was as to the nature of the lesion. The absence of the cardinal signs of a cerebral tumour, namely, optic neuritis, vomiting, and marked headache, was suggestive of an infiltrating growth rather than an isolated mass causing vascular disturbance. Treatment by mercurial inunction, and iodide of potassium internally was adopted, but of course without any permanent benefit. The whole duration of the illness from the onset of the first symptom was three months.

We are indebted to Dr. Sidney Gilford for the photographs from which the illustrations are taken.

(*Exhibited March the 18th*, 1904.)

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58. CONGENITAL WORD-BLINDNESS IN CHILDREN.

By SYDNEY STEPHENSON, C.M.

That some children are much slower than others in learning to read must be an observation as old as the art of reading itself. It is, however, only within the last few years that such tardiness in otherwise bright children has been recognised by a special name and studied with some approach to scientific accuracy. The first writer to describe the condition was Dr. W. Pringle Morgan (1). His patient was a lad aged 14 years, who although intelligent in every other way, experienced the utmost difficulty in learning how to read. The defect was so marked that despite seven years' constant application, he was able to spell out only monosyllabic words. Figures, curiously

enough, he could read fluently, and simple arithmetic sums were done fairly well. A couple of years after the publication of Dr. Morgan's modest communication Dr. Bastian (2) described a similar case in a young man, aged 18 years. This patient was so extremely slow in learning to read that his tutor almost despaired of ever being able to teach him. Dr. James Hinshelwood (3), of Glasgow, next took up the subject, analysed the cases previously reported, and added two new examples to the growing list. To Dr. Hinshelwood undoubtedly belongs the credit of having been the first to describe the condition scientifically, to assign it to a congenital defect in the visual memory centre for words and letters, and to name it "congenital word-blindness." It is highly probable that but for Dr. Hinshelwood's article the two previous communications would have failed to attract the attention they have since received. Mr. Nettleship (4) next described five cases, and in doing so spoke of having been familiar with the condition for many years, thinking and speaking of it as a congenital want of power to acquire knowledge by printed signs. In the year 1902 Dr. Hinshelwood (5) reported two further cases, and took the opportunity of elaborating several of his previous arguments with regard to the essential nature and symptoms of the condition. The other reported cases, up to the date of writing, include one by Lechner (6) and two by Wernicke (7).*

So far, then, fourteen cases of congenital word-blindness have been reported by six authors. To conclude, nevertheless, that this congenital defect is rare on account of the small number of cases reported in literature would be to commit a logical fallacy. Almost

* Since the above was written, I find that Dr. James Kerr presented three males, aged respectively 9, 11, and 19 years, suffering from congenital word-blindness, to the Bradford Medico-Chirurgical Society, on April the 24th, 1900 (see 'Lancet,' May the 19th, 1900). The same author, indeed, had noted the occurrence of such cases four years previously, in a prize essay of the Royal Statistical Society.

everybody who has written on the subject, notably Dr. Hinshelwood, has commented upon the fact that cases are not so rare as the scarcity of records might lead one to infer. Slight cases are almost certain to be overlooked in the hospital out-patient room, where surgeons are busily occupied and parents seldom capable of giving a clear account of their children's ailments. On the other hand, congenital word-blindness, especially when of slight degree, is much more likely to be recognised in the consulting-room, and, as a matter of fact, most of the reported cases appear to have been met with in that way. At the same time, there is no present ground for the belief that the condition is commoner in children of the poorer classes. One point should be mentioned, namely, that 85 per cent. of the cases have been reported by ophthalmic surgeons. This observation goes to show that patients, in the first instance, at any rate, generally seek special rather than general medical advice.

The subject of word-blindness is interesting both from the medical and from the educational standpoint, a fact that leads me to place on record two instances—one slight and the other severe—that have fallen under my notice. The facts are as follows:

Case 1.—Roger B—, aged 9 years, was seen on December the 10th, 1898. Although a painstaking boy, he had experienced unusual difficulty in learning to spell and to read. Arithmetic, also, caused him unusual difficulty. He complained that his eyes got sore after doing his lessons. He had considerable powers of observation and of insight. For example, he could carry in his mind the details of a machine or of a building, and could reproduce them on paper. He reasoned well. In disposition he was considerate and sympathetic. His father, an exceptionally well-cultured man, believed that his son's failure to read was due not to a mental but to a developmental defect. Master B— was of a nervous temperament, but was tolerably well grown for his age and apparently in good health. His relative visual

acuity was almost normal (V. = $\frac{6}{6}$ iij and No. 1 Jaeger), but he deciphered the letters on the Snellen distant type in a curiously hesitating way, a difficulty that extended both to the large and smaller letters. The small types of Jaeger were read slowly and with many mistakes, but what struck me as peculiar was that the larger type on the same test was read with just as much difficulty. Under a cycloplegic: R.V. = $\frac{6}{18}$ + 1·5 sph. = $\frac{6}{6}$; L.V. = $\frac{6}{12}$ ij + 1·5 sph. = $\frac{6}{6}$. Fundi normal; fields of vision for white full; colour sense not noted. Spectacles (1·0 D. spherical) were prescribed for reading, and the father was advised to educate the lad privately. When I saw the patient again some six months later, I found that he had made considerable progress with his reading. The boy died from influenzal peritonitis on January the 15th, 1904—that is to say, some five years after he first came under my notice. According to information kindly furnished by his father, distinct progress had been made in the last year of life. His spelling had improved, especially of Latin words, a language that is more phonetic than English. The improvement had extended to reading and to arithmetic. He was learning to draw from the solid, and was doing it well. "I am exceedingly glad," the father wrote, "that, following your advice, I did not send him to school. Now that the little fellow is gone, it would have been a life-long regret to me if I had embittered his last year of life by placing him where he could not have learned without pain."

Case 2.—John B—, aged 10 years, was seen by me on January the 15th, 1903. This excitable lad had been affected for about five years with spasmodic movements of the face, eyelids, mouth, and neck, especially marked when under the influence of emotion, with a so-called "habit spasm," in fact. He was brought because he had experienced great difficulty in learning to read, and was believed on that account to be "short-sighted." He had never suffered from scarlet fever, rheumatism, or "growing pains." His heart sounds were normal, and

he was not subject to sore throat, such as many rheumatic children are affected with. He was deaf (watch close to ear) on the right side, owing to long-standing otorrhœa.

Family history.—Great grandmother died at 75 years from a third stroke, and she was paralysed down one side for many years before her death. Mother, maternal grandmother, and younger sister have all been affected with rheumatism, the last-named after an attack of scarlet fever. His eldest sister, now 13 years of age, read well when she was eight years old. Father and mother both came from large families, but no reading defect amongst the members has ever been heard of. No insanity or hysteria or "fits." No miscarriages or still-births.

Upon examining the lad, sight was found to be R. $= \frac{6}{36}$ and L. $= \frac{6}{24}$. Atropine was prescribed, and a few days later his refraction was estimated, with the following results:—R. V. $= \frac{6}{60} \frac{+ 1{\cdot}50}{+ 3{\cdot}50\ 90^\circ} \frac{6}{12}$; L. V. $= \frac{6}{36} + 4{\cdot}0\ 90^\circ = \frac{6}{12}$ iij. Fundi normal. Glasses were prescribed for constant wear.

About a year later (January 13th, 1904) I obtained a much more complete account of the patient's tastes, peculiarities, and defects. He was described as being "as bright as a lark and as sharp as a needle." He pondered over books, but did not enjoy them, owing to the difficulty of reading them. Handwriting was read as badly as printed matter, and figures were read as imperfectly as either. Dictation was badly done. Although unable to express himself in writing, yet he can copy sentences from a printed book correctly. He is almost unable to learn by heart from a printed page, but he can memorise quickly and retain tenaciously anything told or read to him. Thus, he learned by rote fourteen verses, each containing four lines, in two days, and repeated them to me without hesitation or mistake. His memory is excellent. At such games as cricket, football, draughts, and dominoes he plays well.

The lad, before he came under my notice, was educated

at a boarding-school at the seaside, and an inspection of his term reports brought to light certain interesting points. From the earlier reports it seems evident that his teachers had no idea of the lad's peculiarity, for they characterised him as "inclined to be idle" (Christmas, 1901). In summer, 1902, we find that "he has made especial progress in arithmetic," although it is to be noted that he obtained only 20 per cent. of the total available marks in that subject. A similar comment with regard to arithmetic is made in the report for the term ended December the 17th, 1903. In short, the boy's strong subjects were Divinity, history, and geography, in all of which he learned the work presumably by the exercise of a very active auditory memory.

Comments.—It cannot be doubted, then, that there exists a congenital condition, not necessarily associated with any other defect (mental or physical), where the learning of letters or of words from printed or written character is difficult or, it may be, even impossible. This congenital letter- or word-blindness may be due, as Drs. Hinshelwood and Lechner surmise, to a congenital defect in the visual memory centre for words and letters. The fact is probably not without significance that of the total number of cases, thirteen—that is, 81·25 per cent.—have been met with in males. The ages of the patients (given in fifteen instances) ranged from seven to twenty-three years, and averaged thirteen years. In other words, they usually fell under notice at an age when the nature of their peculiarity would be forced upon the attention of parents and teachers. A neuropathic family history was forthcoming once only, viz., in one of Wernicke's cases, where the father, an intemperate man, died insane, and the patient's elder brother was weak-minded. It may be noted in passing that another brother could play several games of chess simultaneously, and could also play without seeing the chess-board. The two brothers of one of Nettleship's patients were stammerers. Some of the sufferers from alexia have evidently been of a nervous temperament, as witness one of Wernicke's patients, a female, nineteen years of age, said to

be hysterical; Lechner's case in a boy of thirteen years, said to be "very nervous"; and in the two cases described in the present communication. The previous illnesses of the patients offer nothing calling for particular remark. As a rule, they enjoyed good health.

Vision was recorded in ten of the cases, and was normal in all except two. Refraction, specifically mentioned ten times, was emmetropic once, and hypermetropic, with or without astigmatism, in the remaining nine cases. The fundus oculi was examined in six patients with negative results. The fields of vision and the colour-sense were normal in the two cases in which they were investigated.

General intelligence was good in thirteen of the sixteen cases, and may well have been so in the other three cases, but no definite note was made with regard to the point. The ability to read figures existed in seven of the cases, was not noted in seven cases, and was poor in my own cases. In this connection I may just say that several years ago I saw a lad of ten years who experienced extraordinary difficulty in reading figures, without any corresponding difficulty in regard to letters and words. There was a trifling amount of hypermetropia (0·75 D.), but his eyes were otherwise normal. Unfortunately, I saw the patient once only and have failed to trace him since. As to music, mentioned twice only in the series, it was stated that patients experienced no trouble in that respect. The auditory memory appears to have been highly developed in most of the patients. One of Wernicke's patients could speak two languages correctly; a patient of Hinshelwood's concealed his difficulty in reading for a time by learning his primer off by heart; Lechner's patient is stated to have known the contents of his school-books by rote; and my second patient could memorise well and quickly anything told or read to him.

What improvement can be expected in these cases? It will naturally vary according to the original severity of the case and the educational means adopted. It may be considerable. It may, furthermore, take place compara-

tively quickly, as shown by one of Hinshelwood's cases, where a boy who had not been able to read after upwards of four years at school, made very considerable progress in recognising letters by dint of constant practice during seven weeks. The improvement in these cases is usually described as "slow, but satisfactory." Perhaps the most striking case is one mentioned by Nettleship, where a boy who suffered from a marked degree of alexia, eventually learned to read easily, and nineteen years after he was first examined, had become a lawyer. It may be concluded, therefore, that with special training even severe cases of congenital word-blindness are capable of considerable improvement. It is clear, moreover, that the earlier such training is begun, the better are the results likely to be. Hence the importance of recognising the defect at as early an age as possible.

The diagnosis offers few difficulties if once the fact be firmly grasped that there is such a thing as congenital inability to read. At least, difficulty would be likely to arise only when the word-blindness co-existed, as in my second case, with a considerable error of refraction. Even under those circumstances the history should guard against error, while the fact that the patient experienced exactly the same difficulty in reading large as he did small print would strongly suggest the presence of this curious congenital defect. It is interesting to add that more than one of the case-histories comment upon the fact that the patient never read for pleasure.

In view of the particular attention now being paid to degeneracy amongst our poorer school children, it is pretty safe to predict that congenital alexia will be found to be commoner than hitherto suspected, even by the medical profession.

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39. NOTES ON A CASE OF RENAL CALCULUS: NEPHROLITHOTOMY.

By J. PORTER PARKINSON, M.D.,

AND

DOUGLAS DREW, B.S.

In bringing this case before the Society, we do so, not because the symptoms of stone in the kidney in children present any characteristic differences from those associated with the disease in the adult, but rather on account of its comparative rarity in children, and also as this case presents certain rather unusual features.

T. M—, a boy aged 9½ years, was admitted into the North-Eastern Hospital for Children, under the care of Dr. Parkinson, on August the 21st, 1902.

The following history was obtained:—Thirteen months previously the mother noticed that the child was passing blood in the urine; at the time the child appeared perfectly well. From that date onwards blood was passed in the urine at intervals, but no pains were complained of with the exception of a "griping pain at the anus," which did not appear to have any relation to the attacks of hæmaturia, although it commenced shortly after the first attack, and was present at intervals up to about two months before he was admitted to the hospital. At no time had the mother noticed the passage of any calculus.

State on admission.—The child looked healthy and was well nourished. The urine, which was acid, contained a

large quantity of blood and some granular casts. This attack of hæmaturia had lasted a fortnight. During this period the child had played about as usual, and did not present any symptoms of renal calculus except the hæmaturia.

On examination of the abdomen there was some rigidity of the muscles on the left side, and on deep pressure under the kidney there was at times slight tenderness, but the kidney could not be felt. The child was kept quietly in bed, and the blood rapidly disappeared, but on allowing the child to get up and play about the ward it reappeared.

A skiagraph, which unfortunately has been mislaid, gave clear indications of the presence of a stone in the left kidney, although the shadow was not sharply defined or very intense.

Mr. Drew saw the case in consultation with Dr. Parkinson, and decided to cut down on the left kidney. This was done on September the 20th, 1902, through an oblique incision in the loin. The kidney was exposed and dislocated on to the loin, where it was carefully palpated, but, as is so frequently the case unless the stone is of large size, nothing could be felt.

The organ was split open along its convex border and the finger introduced into the pelvis, where the main calculus was felt and removed; many other minute calculi were felt, many of which were concealed in the recesses of the calyces, and it required the most careful attention to avoid overlooking some of these minute stones; to ensure that none had escaped removal the calyces were washed out with a strong stream of hot water, which also served the useful purpose of checking the free oozing from the incised cortex. A probe was passed down the ureter into the bladder, but nothing abnormal was felt. The kidney was then stitched up with catgut and returned, and the wound was closed in layers by silkworm gut, a drainage-tube being inserted down to the kidney; the tube was removed on the fourth day.

Considerable shock developed during the later stages of

the operation, and persisted for some hours after the child was returned to bed.

During the first four days after the operation the urine, which was copious, was deeply stained with blood.

The child made an uneventful recovery, the wound healing by first intention.

The child was examined on January the 27th, 1904, and found to be in excellent health, and had had no return of the symptoms.

Characters of the calculi.—Sixty-seven calculi were removed; they were all of dark brown colour, irregularly rounded, with a smooth surface, and composed of uric acid, the largest measuring about half an inch in its longest diameter, and the others varying in size from that of dust shot to a hemp seed.

Remarks.—That stone in the kidney in children is very uncommon is evidenced by the following facts.

Ashby and Wright state in the last edition of their work on diseases of children that they have only performed nephro-lithotomy on two or three occasions.

Only one other case has occurred at the North-Eastern Hospital for Children since 1880, when the operation was first introduced. This case was operated upon successfully by Mr. Bilton Pollard.

These facts are somewhat surprising, considering the comparative frequency of vesical calculus in children. It would be interesting to know the ratio that renal calculus bears to vesical calculus both in the male child and the male adult. Doubtless it would be found to be very much smaller in children than in the adult.

Another condition that does not appear to influence the frequency of this disease is the frequent occurrence of the so-called uric acid infarcts in the kidneys of newly-born babies. Holt remarks on this fact and points out that renal calculus is extremely rare in infants.

The unusual features in this case, to which we would invite attention, are:

1. The profuse hæmaturia and its constant relation to

the taking of exercise. Hæmorrhage is not usually a marked symptom, unless the calculus is of large size and very rough, whereas in this case the stones were small and smooth.

2. The absence of all other urinary symptoms except such slight tenderness that it was of doubtful significance. If the skiagraph had not indicated which kidney was at fault there would have been considerable doubt and the operation would have been purely of an exploratory nature. The ill-defined shadow in the skiagraph is explained by the composition (uric acid) and characters of the calculi.

3. Although more calculi than one are often present, yet it is most exceptional to find them in such large numbers (sixty-seven removed).

4. It might have been expected that some of these minute calculi would have escaped down the ureter, and would have been found in the urine, but this did not occur while the child was under observation in the hospital, and the mother had noticed nothing of the kind. Moreover if it had occurred it would probably have been associated with some pain or discomfort, even if the calculus was not large enough to cause actual colic.

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60. CASE OF CONGENITAL PROPTOSIS AND MENINGOCELE.

By JAMES TAYLOR, M.D.

C. B—, aged 5½ years, the eldest of a family of three. The two other children are healthy. There were no miscarriages. The patient was always peculiar in appearance, had always a "soft place" at the back of his head, and his eyes were prominent.

He now presents a markedly angular skull, very prominent eyes, distinct nystagmus, and his vision has lately become, his mother says, much impaired. He has

atrophic discs. There is also an occipital meningocele. The mental condition is defective, but not so much so as his appearance would lead one to expect.

(*Exhibited April the 15th*, 1904.)

Discussion.

Mr. Sydney Stephenson said Dr. Taylor's case seemed to be a typical instance of the uncommon condition known as oxycephaly, several cases of which had been lately published in this country. Only the previous day he had the good fortune to meet with a marked case in a boy aged 17 years. In all the cases which he had been able to examine carefully there had been pallor of the optic discs, which seemed to be secondary to neuritis. It was an interesting study why those steeple-skulled children should develop neuritis, and he would like to know how Dr. Taylor explained the pallor in his own case. He presumed the neuritis was probably due to a mechanical factor, just as the proptosis and divergence of one eye, which were constant features of such cases, were also due to that factor. The explanation was probably to be found in the abnormal shape of the orbit.

Dr. James Taylor, in reply, asked whether Mr. Sydney Stephenson, in his own experience, had found in such cases the condition of meningocele. It was strange that neuritis should have been present from a mechanical cause, while a safety-valve existed in the shape of the meningocele.

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61. MENINGEAL SYMPTOMS OCCURRING IN THE COURSE OF MEASLES—? MENINGITIS.

By JOHN ANDERSON SMITH, M.D.

(Introduced.)

The following case seems worthy of record, as it illustrates a complication of measles which is very rarely seen. The patient, a girl aged 5½ years, was first seen on February the 25th, 1904, when she presented a typical measles rash, cough, slight bronchial catarrh, and a temperature of 103° F. The child did not seem ill, and beyond the fact that the temperature was rather high, reaching 104·2° F., there was nothing to distinguish the

attack from an ordinary moderately severe case of measles. The rash was fully developed and pursued the usual course. On February the 28th, whilst the rash was still out, the child became semi-conscious, vomited three or four times, developed a squint, and retraction of the head. She could not bear the light, and lay on her side away from the light, with her legs curled up. Incontinence of urine and fæces was present. The head became more retracted, and whenever touched she groaned as if some hyperæsthesia were present. She wasted rapidly during the next ten days, and was completely aphasic. The pupils were equal and dilated. There was no otorrhœa, but the mother said there had been a little discharge from the right ear the night before the meningeal symptoms set in. The measles rash faded in a normal manner and the temperature did not seem materially affected. There was no optic neuritis. Kernig's symptom was not present. There were no convulsions at any time. Lumbar puncture was not tried.

March the 22nd.—Aphasia is still absolute; the child lies on her back and cannot move her lower limbs. The pupils are equal and act to light; no nystagmus. Some twitching of the facial muscles. Marked inco-ordination of arms and occasional athetoid movements of fingers. The child appears to understand all that is said; can protrude tongue and can swallow. There is no mastoid tenderness and nothing abnormal in the ears. There is slight general rigidity. The knee-jerks are much exaggerated and ankle clonus is present. No Babinski reflex can be obtained. There is general anæsthesia to pin-pricks, but she recognises hot and cold and appears to fear pinching. No bed-sores.

March the 26th.—Some efforts at speech and putting on flesh.

April the 2nd.—Can sit up without help, and further improvement in speech.

April the 6th.—Anæsthesia less marked, and general condition improving.

April the 23rd.—Has complete control over bladder

and rectum; speech much better; reflexes still much exaggerated, but less anæsthesia; makes some attempt at walking when supported, but cannot stand alone, and considerable ataxia on moving limbs.

May the 25th.—Can stand against the bed but not alone. Has put on nearly 2 lbs. in weight. Able to feed herself and speak. For about a fortnight she became sulky and morose and would not attempt to do anything, but is now happy and ready to try and do what she is told.

There were never any localising symptoms and I think cerebral abscess can be excluded. The duration and gravity of the symptoms seem to point to something more than mere toxæmia and suggest rather a cerebro-spinal meningitis. There was no doubt as to the original attack being measles, and not the rash of a cerebro-spinal meningitis: further evidence of this was seen in the fact that the mother (the only member of the family who had never had measles) developed measles fourteen days later. There was no tubercular history in the family and the child had never been ill before. Meningitis coming on after measles as a sequel, especially in tubercular families, is well known, but the development of meningeal symptoms during and at the height of measles is sufficiently rare to deserve record. Sir T. Barlow records a case of fatal myelitis, with an autopsy, occurring in a young adult in the course of measles where the exciting cause seemed to be the toxin of measles, and von Starck reports a case of spinal meningitis in a child aged 8 years due to measles.

My patient has certainly improved very much, but whether she will ultimately recover completely seems a little doubtful. She is said to have been a bright and intelligent child before the illness.

References.

Von Starck.—'Jahrb. Kinder.,' Leipzig, 1898, N. F. 47.
T. Barlow.—'Lancet,' ii, 919, 1886.

(*Exhibited April the 15th*, 1904)

Discussion.

The Chairman (**Dr. C. O. Hawthorne**) said the only question which occurred to him was whether it was quite justifiable to assume that there was meningitis in this case. The alternative hypothesis would be that the symptoms were due to the development of some form of toxin. He had seen a case having very similar symptoms, and in which complete recovery followed, in an adult after an attack which was described as influenza.

Dr. Farquhar Buzzard thought the symptoms in the case could be explained by a toxic affection of the cerebrum, without the necessity of assuming any lesion in the spinal cord. The anæsthesia, of which only a slight degree now remained, appeared to have been of the cerebral rather than of the spinal cord type, seeing that the superficial reflexes were extremely brisk, whereas the perception of pain seemed to be blunted. The aphasia was also probably of cerebral origin, and the motor paresis could also be accounted for by a cortical lesion. He believed the condition was a form of encephalitis produced by the measles toxin. He thought one seldom got encephalitis without meningitis, and *vice versâ*, but the meningitis in the present case did not appear to have been very marked. As the patient had done so well up to the present, there was reason to hope for a good ultimate result.

Dr. James Taylor said he had never seen a similar case following measles. Some months ago he showed a case which seemed to show very profound effects from some toxic poisoning, not only in the cerebrum but also in the spinal cord and peripheral nerves. The suggestion of the Chairman as to toxæmia being at the bottom of the condition, which Dr. Anderson Smith seemed inclined to adopt, was probably the correct one. With the toxæmia he felt no doubt that there were certain possibly slight structural alterations. But the hopeful feature of such cases was that the structural alterations frequently passed away, and he agreed with Dr. Buzzard in thinking that the child would probably recover ultimately.

Dr. G. A. Sutherland said he had never seen such symptoms arise after measles. With regard to the toxæmic theory, there seemed to be present rather more than general toxic poisoning as in diphtheria, because in the present case the symptoms had come on during the acute stage of the illness—namely, on the third day; usually toxic symptoms were of slower development and came on later in the disease. He agreed with Dr. Buzzard that it was difficult to draw the line between cortical encephalitis and meningitis, and he thought that in the present case one might assume that there was an active meningitis and encephalitis going on at the same time. Considering the severe nature

of the lesion, he scarcely thought recovery would be complete. The present case was one of great rarity and interest, and it would be very valuable to hear the sequel.

Dr. John Anderson Smith (introduced), in reply, said he thought the symptoms were a little more severe than would be accounted for by toxæmia, and that, as Dr. Taylor suggested, there was at present, or had been, some structural change. At the onset the symptoms appeared to be those of meningitis, and he thought the child would not recover. With regard to the possibility that it might not have been measles, but cerebro-spinal rash, the only other member of the family who had not had measles before developed that disease after the onset in the present patient, and that seemed to corroborate the diagnosis.

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62. TWO CASES OF MICROPHTHALMOS, ONE SLIGHT AND THE OTHER EXTREME.

By SYDNEY STEPENSON, C.M.

Case 1.—Albert W—, aged 6 years, first seen at the North-Eastern Hospital for Children on March the 31st, 1904. The child is moderately intelligent, and, save for his eyes, presents no obvious deformity beyond preputial adhesions and a poorly-marked post-anal dimple. His face and legs are deeply pitted from a recent attack of smallpox. Height, 37½ in.; weight, 37½ lbs. A small convergent squint of the left eye is present. The eyes and eyelids are obviously small. The palpebral fissure on each side is 23 mm. in length and 6 mm. in height. The transverse diameter of each cornea measures 8 mm. There is fairly rapid lateral nystagmus, which practically ceases when the lad looks strongly upwards or downwards. There are traces of connective tissue about the central vessels on each optic disc. No coloboma of the choroid or other coarse fundus change. Refraction (estimated under eumydrine) :—

$$\text{R. E.: } \frac{+3{\cdot}0 \text{ sph.}}{+2{\cdot}0 \text{ cyl. } 90^{\circ}}; \quad \text{L. E.: } \frac{+8{\cdot}0 \text{ sph.}}{+1{\cdot}0 \text{ cyl. } 90^{\circ}}.$$

Case 2.—Muriel E—, aged 1 year and 4 months, first seen at the Evelina Hospital on March the 22nd, 1904.

The baby had been born at term, after a labour lasting two days, without instruments. She was born apparently without eyes. There is no family history of deformities.

The normal position of the eyes is marked by a depression, just as if the eyes had been removed surgically.

FIG. 16.

No facial paralysis. Eye-brows, nose, and neighbouring parts well formed. The palpebral fissure on each side measures 5 mm. in length. Upper cilia thick and numerous; lower cilia finer and more scanty. Tears run over the cheek whenever the baby cries, which she does during the whole of the examination. Upon deep palpation through the upper eyelids a small body, resembling a pea in size and shape, can be felt in each orbit. It is difficult

to separate the eyelids, but when this is done by a small speculum, mucous membrane can be recognised, but nothing more.

Remarks.—The two foregoing cases, although at first sight they appear to be very different, are both, I think, examples of microphthalmos. The elder child shows merely unusually small eyes and eyelids, lateral nystagmus, convergent squint, and hypermetropic astigmatism of greater degree in one eye than in the other. The associated congenital conditions include preputial adhesions and a post-anal dimple. In the younger child the conditions are very different. The eyebrows, nose, and neighbouring parts are well formed, but the eyes themselves appear to be absent. When the eyelids are separated (by no means an easy thing to do) a mucous membrane can be recognised lining the interior of the palpebral aperture. When the child cries, tears run over the cheek from between the palpebral fissures. Upon deep palpation through the upper lids a small body, like a pea, can be felt towards the back of each orbit. No other deformities are present.

Not many years ago my first case would have been called one of microphthalmos and my second anophthalmos. It is now becoming recognised, however, that both are instances of microphthalmos, although of very different degrees. The term anophthalmos, strictly speaking, should be applied only to cases where no rudiment of the eyeball can be either seen or felt in the orbit, even when the examination is made under a general anæsthetic. Such cases undoubtedly occur, but they are extremely rare. In some of those examined the optic nerve does not enter the orbit or is absent altogether, while in others there is no optic chiasma or tract, or the external geniculate bodies or the optic foramen is missing. There have been one or two instances where the olfactory lobe was absent as an associated lesion. In all likelihood the small body present in the orbit in my second case represents a rudimentary eyeball containing more or less immature nervous elements, and upon that ground I

prefer to regard it as an example of extreme microphthalmos rather than of anophthalmos.

In some cases of extreme microphthalmos more or less delicate cysts have been found to be connected with the ill-developed eyeball. These are interesting from a developmental point of view, since they contain tissue that may be recognised as of retinal origin. This modified retinal tissue is found upon dissection to be anatomically continuous with the folded retina which lines the rudimentary eyeball. No cyst of the kind described can be made out in my second case.

In discussing the question of microphthalmos *versus* anophthalmos, E. T. Collins and J. H. Parsons ('Trans. Ophthal. Soc.,' vol. xxiii, 1903) have recently pointed out that the essential element of an eye is the nervous mechanism, receiving visual sensations and transmitting them to the brain. They suggest that when there is complete failure in the development of this mechanism—as, for example, when the chiasm or the optic nerve is absent—the condition should be regarded as one of anophthalmos. On the other hand, if the eye is smaller than usual, and there is even an imperfect attempt at the formation of the nervous mechanism, the case is to be looked upon as one of microphthalmos. It is unfortunate that this scientific distinction between the two conditions is one that can be established with certainty only by a microscopic examination.

(*Exhibited April the 15th*, 1904.)

63. TWO CASES OF ANÆMIA.

By E. P. BAUMANN, M.D.

The patient, a girl, aged 10 months, had been ailing for six weeks. She was very pale, and presented an enlarged spleen and well-marked syphilitic stigmata. The blood showed a slight degree of leucocytosis, consisting of an increase in the number of lymphocytes at the expense of the polymorphonuclear cells. There was no

relative increase in the number of the eosinophile and basophile cells. Only a few myelocytes were seen in the blood-films. The red blood-corpuscles were pale, and presented slight variations of shape; a few were nucleated.

Red corpuscles	2,884,000 per c.mm.
Leucocytes	10,000 „
Hæmoglobin	30 per cent.
Colour index	0·5

Differential count (500 leucocytes counted):—

Polymorphonuclear	35	
Small mononuclear	54	60
Large „	6	
Eosinophile	2	
Myelocyte	2	
Mastzellen	1	
	100	

(3 nucleated red corpuscles seen to every 100 leucocytes counted).

Whilst admitting that most observers would classify this case under the heading of Anæmia Splenica Infantum, he ventured to consider it one of anæmia secondary to syphilis. The child was obviously syphilitic, and the results of the blood examination revealed the existence simply of a severe secondary anæmia, modified by its existence in a young child. He had followed such cases through their whole course, and even when the patient became extremely anæmic and died the blood never developed the leucocytosis and marked increase of myelocytes and nucleated red corpuscles which is characteristic of splenic anæmia in infants.

The second case was that of a girl, aged 1¼ years, who was said to have grown progressively whiter in appearance ever since an attack of bronchitis seven weeks before. The child was fairly well nourished, but extremely pale, the skin having a greenish-yellow tint. No syphilitic stigmata were evident. The spleen was enlarged. The blood showed a well-marked leucocytosis, the increase consisting of an increase of the mononuclear at the expense

of the polymorphonuclear cells. There was no relative increase in the number of the eosinophile or basophile cells. Only a few myelocytes were seen in the blood-films. The red blood-corpuscles presented striking variations of size and colouring, and a large number (63·5 to every 100 leucocytes counted) were nucleated. A considerable proportion of the nucleated red corpuscles were megaloblasts. Many were multinucleated; in some cases three, four, and even five nuclei were present, and in a number karyokinetic changes were evident.

Red corpuscles . .	960,000 per c.mm.
Leucocytes . . .	35,000 „
Hæmoglobin . . .	15 per cent.
Colour index . . .	0·8

Differential count (700 leucocytes counted) :—

Polymorphonuclear . . .	27	
Small mononuclear . . .	64	70
Large „ . . .	6	
Eosinophile	1	
Myelocyte	1·5	
Mastzellen	0·5	
	100	

Dr. Baumann stated that the variations in size and colour of the red corpuscles, and the large increase in numbers of nucleated red corpuscles were most remarkable. He was inclined to consider the case as one of those rare instances of the occurrence of pernicious anæmia in infants. The blood picture did not in every respect correspond to that of the blood in adult pernicious anæmia, but the fact of its occurrence in childhood might serve to explain the modifications in the characters of the blood changes.

(*Exhibited April the 15th*, 1904.)

Discussion.

Dr. A. E. Jones said that with reference to the young child with a large spleen and a slight degree of anæmia, it seemed to him that one must provisionally call such cases splenic anæmia, because they conformed to the definitions of that disease, *i.e.*

enlarged spleen with moderate anæmia but with no characteristic blood changes. It was possible that syphilis was the cause in the present case, but he thought the spleen was too large for that to be the case, and there was none of the leucocytosis commonly associated with syphilis causing that degree of anæmia. With regard to the second case, he agreed with Dr. Baumann that it would be best to call it the pernicious anæmia of children. The most striking feature in the case was the large number of nucleated red corpuscles. It was true the number of leucocytes was 35,000; but if pernicious anæmia occurred at all in children one would expect a leucocytosis, as that symptom was brought about by slighter causes in children than in adults. The increase in the leucocytes was only in the lymphocytes: it was not a polymorphous leucocytosis. If nucleated red corpuscles occurred with leucocytosis, the condition was nearly always lienomyelogenous leukæmia. However, he remembered a case in which he counted 700 to 800 red corpuscles to the c.mm., although the case was one of lymphatic leukæmia. On the whole, then, he would agree with Dr. Baumann that this was probably a case of pernicious anæmia.

The Chairman (**Dr. C. O. Hawthorne**) said he would accept Dr. Baumann's view with regard to the first case. He had little doubt that enlargement of the spleen might be found in children of syphilitic inheritance without any other sign of specific disease.

Dr. E. P. Baumann, in reply, said that in the course of a large number of examinations of the blood in children he had never met with anything resembling the picture presented by this case. He had once seen a somewhat similar condition, though less marked, in the blood from a case of advanced cancerous cachexia in an adult; here, too, there was a leucocytosis of the mononuclear type.

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64. A CASE OF MULTIPLE PERIOSTITIS.

By ALEXANDER MORISON, M.D.

Dr. Alexander Morison showed a case of multiple periostitis in a female child aged 15 months. He preferred the term "multiple periostitis" to "polydactylitis," inasmuch as the metacarpal bones of the right hand were chiefly affected, with a similar condition to that of one of the phalanges of the left hand. As the child also exhibited two tuberculous nodules in the skin over the right knee, he regarded the

periosteal lesions as in all probability produced by the same disease. There were no constitutional symptoms. The child had improved in all respects under dietetic and medicinal treatment without any local applications or mechanical restraint of the affected parts.

(*Exhibited April the 15th*, 1904.)

Discussion.

Mr. Lockhart Mummery agreed in the belief that it was a tuberculous case, and said that at the present time he had a case almost exactly like it. Both hands were affected like the present case, and there were several subcutaneous nodules in the skin. In his patient there was middle-ear disease, which he thought was tuberculous also. He did not think those cases were satisfactory from a surgical point of view; the amount of mutilation necessary for adequately dealing with the mass under the periosteum was not justified by the results. The line of treatment, he thought, was rather to adopt medical treatment: fresh air and general hygiene. Of course, if there was a tendency to form abscesses the case was different, but there should be no opening made otherwise.

Dr. H. Skelding (Bedford) thought the mere treatment with a splint would be sufficient. Operation in the present case would necessitate the removal of more than one entire digit. The child seemed to be very extensively diseased, but the majority of cases, with care, did greatly improve. He had seen cases of similar character recover completely without operation, and unless one also removed the nodules from the knees, which he did not think would be advisable, there would be no advantage in operation. Certainly treatment by means of a splint was most satisfactory, coupled with careful dieting.

Mr. L. A. Bidwell agreed that rest was a most important factor in the treatment of cases like the present, but he did not entertain the same objection to operation which had been expressed by other speakers. He would not advise removal of bone, merely incision down to the periosteum and scraping the tuberculous area, the wound being closed up without drainage. He had found tuberculous dactylitis cases healed up well after operation, and the duration of treatment was shortened.

Mr. R. C. Dun (Liverpool) said he had also seen several cases similar to the one which had just been shown. It was inadvisable in the present stage to do anything surgically; it was better to delay any such procedure until the disease was obviously approaching the surface. It was important to fix the hands at the present stage of the disease. In all such cases he was in the habit of putting the hand into a splint, and that, associated with

general treatment, often prevented the further development of the process.

Mr. R. P. Rowlands suggested it might possibly be syphilitic periostitis of the bones. The points which were in favour of that view seemed to be the great deepening of the shadow in the skiagram as compared with what one generally saw in tuberculous dactylitis, and the multiplicity of the lesions in this case. Antisyphilitic treatment might be tried at a venture. Certainly operation should be deferred until treatment by fixation had been thoroughly tried, for radical treatment of so many bones would leave the hand useless.

Dr. Alexander Morison, in reply, agreed that if the hand were kept at rest it would be beneficial, and up to the present there had been steady amelioration of the symptoms without this measure being taken. The mother looked carefully after the child, and there seemed to be very little movement in the bones. Syphilis was considered, and there was the notorious difficulty in procuring a history of that disease, but the family history was good; the child was one of two in the family, the other being older and alive. The mother had had no still-births or abortion; she was in good health, and her husband also. There were also two grandparents alive Because of the absence of evidence or the likelihood of syphilitic taint he had not tried anti-syphilitic treatment. The dark shadow regarded as diagnostic by one speaker was the result of deepening of the shadow in the skiagram by a lead pencil.

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65. A CASE OF TUBERCULOUS NODULE IN THE PONTO-MEDULLARY TRACT.

By S. VERE PEARSON, M.B.

Bertram O—, aged 2 years, was brought to me a fortnight ago for weakness and disinclination in walking, with slight drowsiness and squint. There was palsy of nuclear type of the sixth and seventh cranial nerves on the left side, with coldness and œdema of the right foot.

The history of the patient was as follows: family history, negative. Six months ago he was perfectly healthy and could walk quite well. Since then weakness had gradually come on, and though able to stand he had shown disclination to walk, and considerable feebleness in doing so.

For two months he had occasionally had retching, usually in the morning, and sometimes "choking" difficulties in swallowing. Some languor and drowsiness had been noticed, as well as an internal squint of the left eye, for three weeks. He had never had any fits, febrile attacks, or otorrhœa. Some swelling of the dorsum of the right foot had been noticed for a few days.

The state of the patient.—Since the patient first came under observation, just over a fortnight ago, definite right-limbed paresis had made itself evident, and there had been no vomiting or headache. Occasionally swallowing is noticeably difficult, and on two occasions there has been slight regurgitation of milk through the nose. He has been inclined to sweat rather profusely, especially when asleep. The present condition is: He is a well-nourished boy of 2 years, mentally normal. He has long eyelashes, some downy hair on the back between the shoulders, and a somewhat tuberculous aspect. There is nothing abnormal discoverable about the thoracic or abdominal viscera. Paralysis of the left sixth and seventh cranial nerves of lower neuron type is present. The pupils react normally. The paralysis of the face is not complete, but the upper half of the face is involved, and there is marked failure to Faradic stimulation in most of the muscles. Hearing on the left side is probably defective. There is no visible hemiatrophy of the tongue, but the left side appears to be paretic. The condition of the palate is doubtful. On examining the ears, and on ophthalmoscopic examination nothing abnormal can be detected. The patient can move his limbs, but moves the right arm and leg less than the left, and there is definite paresis of these extremities, with coldness and either pallor or blueness, and a tendency to slight œdema of the hand and foot. There is no muscular atrophy, the limbs measuring the same as those on the opposite side. Spasticity is not present. The tendon and plantar reflexes are sluggish and somewhat variable, but appear to be normal, unless there is slight increase of the right knee-jerk. Sensation, as far as it can be tested, is normal. In bed he lies on

either side. He can sit and play with toys, but uses his left hand almost entirely. There is no nystagmus.

Diagnosis.—Symptoms of basal meningitis were not present, and with that lesion a crossed paresis as seen in this case would be unlikely. There were no indications of syphilis. The symptoms would not conform to infantile palsy, nor with a cerebral birth palsy. The age of the patient practically excluded hysteria. There remained therefore for discussion the differentiation between a cerebellar lesion interfering with the muscular power of the limbs and pressing upon the left cranial nerves as they came out from the pons and medulla, and a lesion in the ponto-medullary tract. If a cerebellar lesion was on the left side so as to press on these nerves, it must interfere with limb movements in one of two ways: either it would by destruction of the cerebellar substance give paresis of the limbs on the left side, or by pressure upon the left side of the uncrossed pyramidal tract it would give rise to a spastic paresis or paralysis of the right extremities. But neither of these conditions was present. The lesion must therefore be in the ponto-medullary tract. With respect to the nature of the lesion everything went in favour of a tumour. The kind of tumour must be doubtful; but the slow progress of the symptoms, the age of the patient, the presence of such indications as downy hair on the back, etc., made tubercle the most probable.

(*Exhibited April the 15th*, 1904.)

Discussion.

Dr. Farquhar Buzzard agreed that the tumour was probably of a gliomatous nature, and that it affected the pons. Tuberculous tumours in that region appeared to be very rare, but when met with in the post-mortem room accidentally, as they were sometimes, they did not appear to have produced any symptoms during life. Gliomatous tumours of the pons produced extremely small and variable symptoms in proportion to their size, even though they invaded the structures extensively. He would not be surprised if in the case under discussion the tumour was far more extensive than appeared likely from the symptoms.

The Chairman (Dr. C. O. Hawthorne) said the only comparable experience he could recall was that of a patient with

evidences of tubercle in various parts, who developed third nerve paralysis. This was proved at the post-mortem examination to be due to a nuclear lesion of a tuberculous character.

Dr. Vere Pearson, in reply, said that he had not seen a pontine tumour before, but from a study of that literature on the subject to which he had been able to refer he thought the present case conformed best to a tuberculous tumour. The age of the patient, too, favoured this diagnosis, for under three years of age gliomata were much rarer than tuberculous tumours. At this age he had not personally met with a glioma, whereas he had seen many cases of intracranial tuberculous tumours. The family history was negative both in respect to tubercle and tumours.

Postscript (*October*, 1904).—Post mortem there was a tuberculous mass in the medulla just below the pons. In longitudinal section it just transgressed the middle line and reached to within about a centimetre of the fourth ventricle in depth. It involved the sixth and seventh nerves on the left side at their origin, and caught some of the left pyramidal fibres before their decussation. There was also extensive tuberculous meningitis.

66. A CASE OF PARAMYOCLONUS MULTIPLEX.

By C. O. HAWTHORNE, M.D.

The patient, a boy aged 9 years, displayed most of the characteristic phenomena of the disease. There were frequent, shock-like, clonic, involuntary muscular contractions, to be observed in the face, limbs, and trunk. There was also conspicuous bilateral symmetry in the distribution of these spasmodic contractions, and corresponding parts on opposite sides of the body were affected about the same instant of time. The spasms were most pronounced in the thighs, upper arms, and muscles of the abdominal wall, and were more marked with the patient in the recumbent posture than when he was standing erect. In all these respects the case corresponded to the classical picture of paramyoclonus multiplex. As somewhat special features of the case, attention was drawn to the frequent (two or three times a minute) jerking upwards of both testicles due to symmetrical and isochronous clonic spasms

of the cremaster muscles; also to the affection of the facial muscles, as these in most instances of the disease escape. According to the boy's grandmother the spasms continued during sleep. There was no history of fright or of any other incident suggested by the boy's guardians as the cause of his condition. He was noticed some three years ago to have the habit of "making faces," and on several occasions had been thrown suddenly and unaccountably to the ground. At no time had he suffered from rheumatism or chorea. The family history included the deaths of two brothers from tuberculous meningitis, but no record of epilepsy. A cousin and a sister of the maternal grandmother had, however, been insane.

(*Exhibited April the 15th*, 1904.)

Discussion.

Dr. G. A. Sutherland asked whether the muscular twitchings were increased under the influence of emotion, and also whether Dr. Hawthorne could give any idea as to the origin. Was there any exciting cause other than that suggested by the family history of epilepsy? Was there any cause which might have excited an underlying tendency to the condition?

Dr. C. O. Hawthorne said he could only reply that those who had charge of the boy had no suggestions to make as to the onset of the condition. The child had been healthy and there was no history of fright. His grandmother, with whom he lived, appeared to have regarded the case simply as one of original sin, and to have treated the boy accordingly.

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67. OPAQUE NERVE-FIBRES EXTENDING OVER A LARGE AREA OF THE FUNDUS, INCLUDING THE MACULA.

By C. O. HAWTHORNE, M.D.

The discovery of opaque nerve-fibres in the fundus oculi, though hardly a frequent ophthalmoscopic experience, occurs sufficiently often to merit in ordinary circumstances

no more than a mere word of recognition. The percentage of such cases in relation to the total number of eyes examined is doubtless a very small one, but a routine use of the ophthalmoscope extending over a considerable number of patients can scarcely fail to detect an occasional example. And as the condition, with rare exceptions, has no practical effect on vision, the opportunity is just as

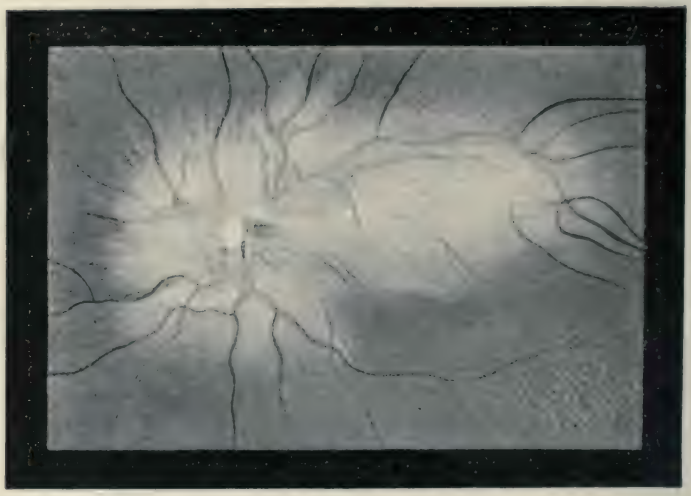

FIG. 17.—Opaque nerve-fibres extending over a large area of the fundus, including the macula.

likely to occur to the physician or other practitioner as it is to the ophthalmic surgeon.

As is well known, the usual arrangement of opaque nerve-fibres in the fundus is in the form of one or more lustrous white patches jutting outwards from the margin of the disc. Possibly by a very critical scrutiny of the visual field it can be shown in some cases that there is a corresponding extension of the normal area of the "blind spot," but otherwise such patches neither produce a subjective sense of impaired vision nor do they qualify the ordinary tests of the visual acuity. Exceptions to the

propositions just stated are so uncommon that the case here figured appears to be worthy of record. In the first place it will be observed (see Fig. 17) that the opaque nerve-fibres are not restricted to one or more isolated patches on the disc circumference, but that, on the contrary, they completely encircle the disc. Further, instead, as is usual, of extending over the fundus from the disc margin for a short distance only, say a disc's breadth or less, the opaque fibres here thrust themselves over so large an area that they form by far the most conspicuous feature in the ophthalmoscopic picture. Especially is this unusual extension pronounced to the outer side of the disc. So extreme is the development in this direction that the opaque fibres are continued over and conceal the site of the macula and also of a considerable portion of the fundus beyond this. It is of interest to observe that, in spite of this highly exaggerated provision of a medullated sheath to the nerve-fibres of the retina, the disc itself, except for a small part of the lower and outer quadrant, is not involved in the process. Another detail worthy of remark is the absence from the opaque area in the macular region of the fibrillated lustrous appearance which is a characteristic of the familiar small patches attached to the disc margin and is also a feature of other portions of the present picture. The macular portion of the opaque area has, on the contrary, rather a "dead" white character and some apparent irregularity of the surface. Of its true nature, however, there can be no doubt. This is suggested by the distinctive character of other portions of the opaque area, by its own relationship to the retinal vessels, and by the striated appearance of its margins. The patient, a girl of moderately dark complexion, had no appreciable structural peculiarity in any other part of the body, and the other (right) eye, except for a slight degree of hypermetropic astigmatism, was in no respect noteworthy. The affected (left) eye, but for the opaque nerve-fibres as above described, appeared normal, though with it there was not even

perception of light, and the pupil light response was very defective. The eye was not divergent, and there was no nystagmus.

Examples of such an extensive development of opaque nerve-fibres as is seen in the case here illustrated (see Fig. 17) would appear to be extremely rare. Some years ago Mr. Hartridge recorded an instance in which the disc was completely surrounded by opaque nerve-fibres with, in addition, a relatively narrow opaque band passing outwards and somewhat downwards for a considerable distance across the fundus. In Jaeger's 'Atlas' also there is a somewhat similar illustration. The instance now reported is more extreme than either of these, both as regards the depth of the investment of the disc and the extent of the invasion of the macular and neighbouring parts of the fundus. It has also this special interest, that the patient's inability to see with the affected eye seems to be entirely due to the extraordinary development of opaque nerve-fibres. (*Exhibited April the 15th*, 1904.)

Discussion.

Dr. George Carpenter said the case was a most exaggerated example of opaque nerve-fibres. He suggested to Dr. Hawthorne that it would be very beneficial to the Society if a black-and-white picture of the case could be made, so that it could be included in the "Reports."

Dr. C. O. Hawthorne replied that he had already made arrangements to supply such a picture.

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68. CHANGES IN THE FUNDUS OCULI FOLLOWING A BLOW.

By C. O. HAWTHORNE, M.D.

This case showed a considerable area of choroido-retinal disturbance extending from the outer margin of

the disc and including the macula, with fine retinal pigmentation. The boy had been struck in the eye with a potato in November, 1903, but suffered so little sense of disability that his parents had never taken medical advice regarding his condition. The affected eye was quite blind.

(*Read April the 15th*, 1904.)

Discussion.

Mr. A. H. Tubby asked whether there was any external strabismus. He had not seen the present case, but had observed three or four in whom there had been loss of vision without loss of the eyeball, and in which the external strabismus had come on at varying periods, from six to twelve months.

Dr. C. O. Hawthorne, in reply, agreed that external strabismus was a common development in a blind eye and might in time possibly occur in the present case.

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69. GONORRHŒAL SALPINGITIS IN A CHILD AGED SIX; REMOVAL OF TUBES.

By LEONARD A. BIDWELL.

On June the 20th, 1902, I was asked by Dr. L. Times to see a little girl aged 6, who was suffering from inflammation of the vulva and painful micturition. The little patient had recently come from South Africa, and there was some suspicion in the parents' minds that she had been assaulted by a Kaffir. Against this view it was noted that the discharge had not been noticed till a month after leaving their home. The child looked rather delicate. There was no elevation of temperature and no enlargement of the inguinal glands. There was some superficial ulceration inside the vulva extending towards the urethral orifice. Examination was very difficult, as the parts were tender. There was considerable pain after micturition, and blood with the urine. I was inclined to think the case

one of simple ulceration, and ordered some simple astringent. On June the 25th I was again asked to see the child on account of a painful swelling in the right foot. There was redness and œdema over the flexor tendon sheath, and pain on movement. The temperature was 102° F. The discharge was about the same. On the following day, as the temperature was still high and the foot was more swollen and the pain more acute, an incision was made and some clear fluid evacuated from the tendon sheath. The wound was closed and dressed antiseptically. At the same time the ulceration in the vulva was mopped with pure carbolic acid, and on further examination it was found that the pus was issuing from the os uteri. The temperature did not fall after this incision, although all the pain was reduced, but on June the 28th the discharge became rather less in quantity and the child complained of abdominal pain. On June the 29th the temperature rose to 103° F., and the patient had severe pain in the abdomen. There was rigidity of the right rectus and swelling to the right of the middle line in the hypogastric region; this was very tender but was not dull on percussion. There was retention of urine. On June the 30th the temperature fell to 100° F., and the child was much better after a thorough evacuation of the bowels. There was less discharge, and the abdominal swelling was less distended. Nothing was felt per rectum. Mr. Watson Cheyne kindly saw the patient with me, and he came to the conclusion that the case was one of gonorrhœal salpingitis, and that it was partially subsiding. An examination of the discharge made by Dr. Eastes showed gonococci in considerable numbers. The child seemed to improve considerably for three days and the pain was much less, and the temperature remained normal till July the 4th, when she again had irritability of the bladder and some pain; the temperature rose to 101° F., the discharge was less in quantity, and the foot was completely well. On the following day the temperature rose to 102° F., and the child was in great pain. On rectal examination the swelling

was felt on the left side of the pelvis, very tender to the touch. On July the 8th the temperature remained up at 102° F., the pain was more severe, and the pulse rate was 120 to the minute; the abdomen was slightly distended and the swelling was distinct.

The patient was restless and refused nourishment. It was, therefore, decided to operate, and on July the 8th the child was anæsthetised by Mr. J. H. Chaldecott, and the abdomen was opened in the middle line above the pubes. There was some slightly purulent fluid in the peritoneal cavity, and some flakes of lymph on the intestines. A dense mass was felt on either side of the pelvis, and on separating adhesions pus escaped: both tubes were found to be full of pus, and so were removed after ligature of their ends close to the uterus. The ovaries were left. The pelvis was sponged out and a rubber drainage-tube inserted. The patient was rather ill for the first few days, but then made an uneventful recovery, and went home on July the 30th with the wound quite healed. The vaginal discharge, however, did not completely cease, and on bacteriological examination it was found to still contain gonococci. The child was, therefore, again put under chloroform on September the 17th, and as the discharge was found to be coming from the uterus, the os was dilated and the uterus curetted. Within a week the discharge had ceased, and I heard some months later that she was in perfect health.

The case presents several points of interest. I believe that gonorrhœal salpingitis is very rare at such an early age, and the course of the disease appears to be rather more acute than in adults. The child was very ill indeed before the operation, and, indeed, I gave a very bad prognosis before operating. I do not think that an operation should be done in such a case until all hope of cure by other means has been abandoned, since the functional result must be sterilisation. I left the ovaries in the hope of avoiding any arrest in the development of the child. It is interesting to note that the discharge did

not stop after salpingectomy, but persisted until the uterus had been curetted. The lesson of the case seems to be that in a case of vaginal discharge which is proved to be gonorrhœal, the patient should be anæsthetised, and if it be found that the discharge is coming from the uterus, the os should be dilated and the uterus curetted; in this way any extension to the tubes should be avoided.

This case is further interesting on account of the acute inflammation of the tendon sheath of the right foot, which was undoubtedly a form of gonorrhœal rheumatism.

(*Read April the* 15*th*, 1904.)

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70. GONORRHŒAL INFLAMMATION OF THE UTERINE APPENDAGES IN A GIRL OF 3½ YEARS DETECTED BY BIMANUAL EXAMINATION; SPONTANEOUS RECOVERY.

By GEORGE CARPENTER, M.D.

Mary W—, aged 3½ years, was brought to the Out-patient Department at the Evelina Hospital on November the 3rd, 1898, for advice as to a vaginal discharge of six weeks' duration; for pains in the lower part of her abdomen, which was not swollen; and for frequency of micturition.

The pus from the vulva contained numerous gonococci.

On a bimanual examination of her pelvic viscera being made by way of the rectum the uterine appendages were found to be involved.

On the right side there was felt an irregularly shaped elastic tumour attached to the uterus at the upper part, and from which it could not be differentiated. Its free extremity was movable. It presented a central depression, and measured, at a guess, 1¼ inches or more lengthwise and ¾ths of an inch across. Rolled between the fingers of the two hands, there seemed to be an ill-defined cord upon it

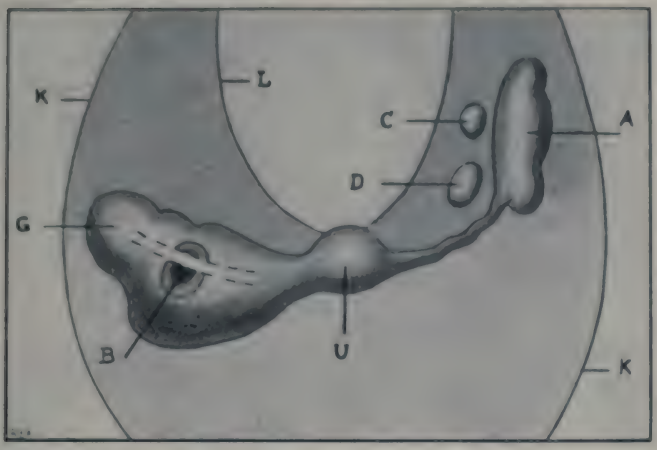

FIG. 18.—Condition of the pelvic genitalia as determined by a bimanual rectal examination on November the 3rd. A. Fusiform swelling in which the Fallopian tube terminated and behind it two rounded bodies D and C. B. Central depression. G. Irregularly shaped elastic tumour with an ill-defined cord upon it shown by the broken line. K. Pelvic brim. L. Falciform ligament. U. Uterus.

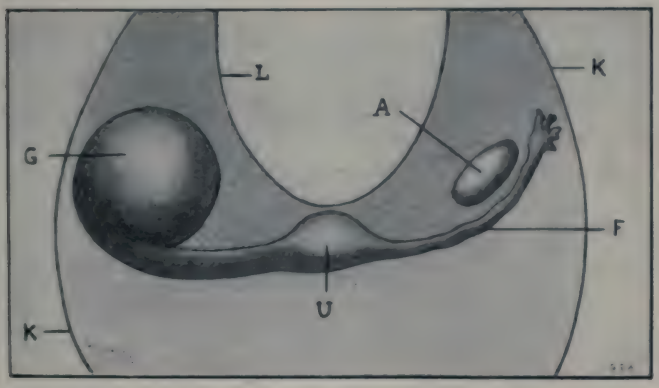

FIG. 19.—Condition of the pelvic genitalia as determined by a bimanual rectal examination on December the 1st. A. Left ovary. F. Left Fallopian tube, both of natural size. G. Elastic tumour on the right side associated with an enlarged Fallopian tube. K. Pelvic brim. L. Falciform ligament. U. Uterus.

at one part, which might or might not have been the Fallopian tube, but if it were so, and that was open to considerable doubt, it was not enlarged. On the left side the Fallopian tube apparently ended in a fusiform swelling, which was attached to the side of the pelvis, and which was about an inch long and a quarter of an inch broad. This was probably the enlarged fimbriated extremity of the Fallopian tube. If it was the ovary, then that organ was decidedly enlarged for her age. Lying below this body were two rounded bodies, somewhat movable, one of them being about the size of a small pea, the other half that in diameter. The uterus felt natural except just where it was attached to the tumour on the right side. All the parts had rather a woolly and indistinct feel, which was thought to be possibly owing to associated pelvic peritonitis.

On December the 1st the pelvic condition had considerably changed. The left ovary and Fallopian tube were found to be of natural size. The right Fallopian tube was decidedly enlarged, and associated with it was a very elastic tumour half an inch or more in diameter. The uterus was then normal to the feel. All parts were freely movable. There was still a purulent discharge from the vagina.

On January the 5th, 1899, the condition of the ovary and the Fallopian tube on the left side remained unchanged. Considerable improvement had taken place on the right side. The Fallopian tube was but slightly larger than natural, and the part in association with it already described, and which might or might not have been the ovary, was also much smaller, but still too large for a healthy ovary. The vaginal discharge was as bad as ever.

On April the 6th the os uteri was examined by a Fergusson's vaginal speculum, which was specially made for the purpose. There was no discharge from it, but the cervical mucous membrane was reddened, and had lost its sheen. The mucous membrane of the vagina was reddened in patches.

She remained under my care until December the 14th,

during which time she passed through attacks of whooping-cough, measles, and varicella.

At the end of May there was a note to the effect that the discharge was less.

There are now a number of cases of localised gonorrhœal peritonitis in little girls on record, but none that I am aware of where bimanual examinations of the internal genitalia have been made during the course of these complications. I therefore record the case as an illustration of pelvic disease secondary to gonorrhœa, and also as an example of the value of bimanual examination of the internal genitalia in young children—a method which I have long advocated, and the technique of which I have described.* There is a further value attached to this case in that it shows that these conditions, at least in some instances, tend to spontaneous recovery, and that without such examinations cases are likely to be passed by. It is interesting to speculate on the effect such complications are likely to produce as regards future child-bearing. Sterility may possibly result in some instances. Marx† is of opinion that these infantile inflammations are apt to commence afresh at puberty, and often are the real cause of pelvic inflammations of newly-married women hitherto frequently credited to the husband.

Lastly, what is the correct interpretation of the information gathered from these bimanual examinations? I am of opinion that there was pelvic peritonitis, and I do not think there is any doubt about there being salpingitis—certainly on the right side and probably on the left. I do not feel quite clear as to whether the rounded tumour in association with the enlarged Fallopian tube on the right side, as described on January the 5th, should be looked upon as an enlarged ovary (oöphoritis) or as a localised distension of the Fallopian tube. If it was the Fallopian tube I did not detect anything at my examina-

* 'Pediatrics,' vol. i, pp. 491—500, "On the Value of Rectal Exploration as an Aid to Diagnosis in Diseases of Children."

† 'Gazette de Gynecologie,' November the 15th, 1895.

tions which was like an ovary. In any event there is no question that the uterine appendages in this case were attacked by gonorrhœal inflammation, and that they apparently spontaneously recovered from it.

(*Read April the* 15*th*, 1904.)

Discussion.

Dr. William Ewart said he regarded both the papers as exceedingly suggestive and of great importance, both from the clinical point of view and from that of medicine in general. The remarks of both Dr. Carpenter and Mr. Bidwell were very valuable, and the Society was much indebted to those gentlemen for contributing the papers.

The Chairman (**Dr. C. O. Hawthorne**) said that one point in Mr. Bidwell's paper which interested him was the complete recovery from the synovitis which had complicated the case. It had been shown on several occasions that the gonorrhœal synovitis which occurred as an occasional complication of ophthalmia neonatorum usually made a complete and rapid recovery. Two years ago he asked the attention of the Society to a case in which this was illustrated, and he then quoted other cases which were put upon record by Mr. Clement Lucas, who was the first to describe the condition.

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71. A CASE OF POST-HEMIPLEGIC CHOREA PETIT-MAL AND MENTAL IRRITABILITY TREATED SURGICALLY.

By R. C. DUN, C.M.

The case which I report is that of a boy aged 4 years at the time when he first came under my care. The history was that when twelve months old a severe convulsion ushered in an attack of pneumonia; during the first few days of this illness several less severe convulsions occurred, and the child was said to have been unconscious. When these acute symptoms subsided it was noticed that the right side of the face and the right upper and lower extremities were paralysed. The boy

had previously been perfectly healthy. In six months' time, under a course of douching, massage, and electricity, the paralysis began to improve. As this occurred it was noticed that the right upper extremity, particularly the hand, and to a lesser extent the right lower extremity, began to be involuntarily moved in a purposeless manner. This condition gradually became aggravated as regards the hand, until it was constantly in motion and practically useless.

About the same time the child began to have "fainting fits," which from the description given me by the doctor in attendance, and from what I afterwards saw of them myself, were undoubtedly epileptic seizures of the petit-mal type. The fits increased in frequency, ten or twelve sometimes occurring in twenty-four hours. Occasionally the boy seemed to have warning of their oncoming, beginning to cry and putting his hand up to the left side of his head.

There was no history of headache or vomiting.

The mental state gradually deteriorated after the onset of the fits. The child ceased to take notice of, or interest in, anything; he spoke less than he had done previously, became restless, difficult to manage, and dirty in his habits.

When about three years old, marked destructiveness appeared, the boy throwing his toys about and tearing up books and papers. Attacks of uncontrollable temper were the worst feature. They came on suddenly, without apparent cause or warning, and during them the boy would fight with his mother or nurse, and attack the two other children with all the power at his command.

Three months before I saw the child he was knocked down by a bicycle, the handle striking him on the left temple. After this accident the fits of temper became more frequent.

There was no family history of nervous disease or syphilis.

The boy had all along received the most careful

general and medical treatment, and had been given prolonged courses of bromides without any improvement in his condition.

When I saw the child he appeared to be well formed and exhibited none of the stigmata of degeneration. The signs of mental deficiency were obvious. He was restless and constantly grimacing, with the saliva trickling from the corners of his mouth. It was difficult to attract his attention, and he resented being touched. The right hand was in constant choreiform motion, and the right leg was similarly affected in a lesser degree.

The muscular power appeared to be equal on the two sides, though there was slight atrophy of the right upper extremities. No spasticity was present, but the right reflexes were somewhat exaggerated. Walking was a little unsteady, apparently owing to the involuntary movements in the right leg. The sight and hearing appeared to be normal, and no optic neuritis was present.

The child spoke little and very indistinctly, muttering to himself and slurring his words. He appeared to understand when spoken to.

A number of the teeth were decayed, and a slight degree of stomatitis was present.

The parents explained that they could no longer keep the child at home owing to his violent temper and the danger of his attacks upon the other children. They were anxious to know if surgical treatment was likely to improve the condition before deciding to send the boy to some suitable institution.

I felt that the chance of improvement following an operation was very slight, and suggested that the teeth should be attended to, in the hope that the removal of an obvious source of reflex irritation might lead to some change for the better.

Seven weeks later I saw the child again; his condition was unaltered. The parents strongly urged operation, and, after warning them that little was to be expected from it, I agreed to their request.

I trephined the child on the left side over the lower part of the fissure of Rolando, and removed bone upwards and backwards corresponding to the lower two thirds of the motor area.

The dura mater appeared to be normal, bulged considerably, and pulsated feebly. On incising it subarachnoid tissue protruded into the opening. It showed an œdematous condition, appeared to be about a quarter of an inch in thickness, and clear colourless fluid escaped on puncture.

The dura mater was freely opened and a widespread œdema of the subarachnoid tissue disclosed, the limits of which were not reached even after a further removal of bone. No definite cyst was present; the meshes of the subarachnoid tissue appeared to be distended with fluid.

Each incision made into the œdematous tissue was followed by the escape of a few drops of fluid and the collapse of a small area of the swollen membrane. In all from three to four drachms of fluid were evacuated.

The subjacent brain appeared perfectly normal. The dura mater was sutured, the majority of the bone removed was not replaced, and the wound closed without drainage.

The after course of the case has been briefly as follows: The involuntary movements of the right extremities and the general restlessness were never noticed after recovery from the anæsthetic. On the tenth day the child began to feed himself with the right hand. His mental condition showed considerable improvement about the same time; he began to pay attention to his surroundings and smiled intelligently at his nurses. The outbursts of temper were less frequent than before the operation, and when spoken to he was able to control them. No fits were noticed during the first month.

I saw the boy a few days ago—six months after the operation. He was bright in appearance, and passed muster as regards intelligence. He spoke rationally and fairly distinctly, and could repeat several nursery rhymes. He now plays naturally with his brother and sister, and is

said to be as easy to manage as they are. The right hand is used very nearly as much as the left, and the boy is cleanly in his habits. He has had eight or ten fits during the past five months.

I record this case on account of the satisfactory results which have followed the treatment. I operated with very little hope of improving the child's condition, and I should not have interfered but for the pressure which was brought to bear upon me by the parents.

Although I feel that it is only rarely that marked benefit can be expected to follow the surgical treatment of such cases, still, considering the slight risks involved, I shall in the future be less inclined to withhold operation than I have been in the past.

(*Read April the 15th*, 1904.)

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72. DELAYED CHLOROFORM POISONING.

By HAROLD J. STILES, C.M.,

AND

STUART McDONALD, M.B.

(Introduced.)

The interest of one of us in delayed chloroform poisoning was first aroused by Dr. Leonard Guthrie, a fellow-member of this Society. Although the occurrence of delayed chloroform poisoning had been recognised in Germany, we are indebted to Dr. Guthrie for directing attention to the subject in this country. His first paper (1) on "Some Fatal After-Effects of Chloroform on Children," published in the 'Lancet' ten years ago, was at once recalled to my (H. J. S.) mind by a case which occurred in the Royal Edinburgh Hospital for Sick Children in 1899, soon after I became surgeon to that institution. So little reference is made to delayed chloro-

form poisoning in English medical literature that it is necessary before recording our own observations to give a chronological review of the work already done on the subject.

Casper (2), in 1850, was the first to direct attention to the dangerous after-effects of chloroform. He maintained that chloroform may exert a poisonous action on the organism hours, days, or even weeks after its inhalation, and speaks of the condition as chronic chloroform poisoning. The first case which he regarded as of this nature was that of a woman who died nine days after amputation of the leg with symptoms referable mainly to the brain.

Casper's teaching was accepted by Langenbeck (3), who, in the same year, ascribed a death seventeen hours after operation to the after-action of chloroform. The patient, a man aged 36, had had his scapula excised. The operation lasted three quarters of an hour. The patient's condition was very satisfactory until the evening of the operation, when vomiting set in and persisted all night. The following morning he was pale, the pulse was small and frequent, and the vomiting continued. The respirations were free and regular, but the pulse became progressively more rapid and feeble, and the patient died at 8.30 p.m. the day after the operation. At the post mortem the liver was found to be very fatty. No further evidence in support of the views of Casper and Langenbeck were brought forward until 1866, when Nothnagel (4) published the results of his experiments on rabbits. In some of the animals chloroform was introduced into the stomach, and in others into the subcutaneous tissue of the back. On examining the organs some days later, marked fatty degeneration of the liver and heart was found in both instances. When introduced subcutaneously, fatty degeneration of the kidneys was also produced, and the urine which was expressed from the bladder four hours after the injection was found to contain red blood-corpuscles and fibrinous casts. In one of his experiments Nothnagel removed a piece of liver, for the purpose of

comparison, before injecting the chloroform. He came to the conclusion that the fatal action of chloroform was not due to pre-existing disease in the affected organs. He favoured the view that chloroform produced fatty degeneration by directly destroying the red blood-corpuscles, and compared its action with that of the bile acids, arsenic and phosphorus. Similar experiments were performed with ether, which produced only a very slight fatty change.

In 1883 Ungar and Junkers (5) were the first to demonstrate in animals the fatty change produced by chloroform when adminstered by inhalation. In the first set of experiments rabbits were employed, but it was found difficult to keep those animals continuously under the influence of the anæsthetic for any great length of time without a fatal result. It was therefore found necessary to interrupt the administration of the chloroform from time to time. In two experiments in which the animals died unexpectedly after the anæsthesia had been kept up 70 and 90 minutes respectively, even microscopic examination failed to reveal any fatty degeneration. In their experiments Ungar and Junkers succeeded in producing only a slight fatty change in the heart. They attributed this to the fact that, in rabbits, they were unable to maintain a sufficient amount of the drug in the blood without producing an immediately fatal result. It is evident, however, from the results obtained by ourselves, as well as by other observers, that they killed the animals too soon after the administration of the chloroform, and before the fatty degeneration had time to develop. In a second series of experiments dogs were used, as it was found that in these animals anæsthesia can be kept up for much longer periods, with less danger of causing sudden death. Ungar and Junkers were able to demonstrate extensive fatty change in the liver, heart, and voluntary muscles in an animal killed forty-eight hours after a single prolonged administration of chloroform. By repeated administrations, in addition to the changes in the liver and heart, they were able to produce fatty degeneration

in the kidneys, the spleen, and in the mucous membranes of the respiratory and alimentary tracts. Ungar and Junkers, after finding that a certain amount of fat was normally present in the convoluted and straight tubules of the kidney in dogs, excised one of these organs, and found that it contained much less fat than its fellow, which was not removed until after the anæsthesia had been continued for a much longer time. In one experiment there was produced a degree of fatty degeneration such as is rarely met with in phosphorus or arsenic poisoning. It is worthy of note that they were able to demonstrate fatty degeneration of the heart in one animal dying suddenly at the commencement of a second administration as early as twenty-four hours after the first, which had been kept up for two hours. In another experiment, in which the interval between the first and second administration was fifteen hours, the animal died during the second day after the second administration, which was of comparatively short duration. The animal had apparently quite recovered from the first, but the authors point out that no doubt the fatty change set up by the first administration was such as to enable a comparatively small amount of chloroform to prove fatal on the second occasion. They suggest that in anæmic, alcoholic, or cachectic patients, or in those who have suffered from long-continued pyrexia, an ordinary dose of chloroform may prove dangerous by producing a fatty change which would not have occurred had the patient been healthy.

Ungar states that Volkmann and Koenig agree that deaths attributed to shock after prolonged operations may be due to the chloroform. Especially after operation on children, says Volkmann, "I have observed a series of fatal cases, presenting the symptoms of so-called shock, which I attributed to chloroform, while others would attribute them to carbolic poisoning."

One of us, who has performed a large number of major operations on very young children, is satisfied that far too

much is made of shock as a cause of death in children. If care be taken not to poison the child with antiseptics, and if undue loss of blood be prevented, serious operations, including abdominal operations, may be performed on even the youngest infant without fear of death from shock.

In discussing the *modus operandi* of chloroform in producing these fatty changes, Ungar and Junkers state that they are unable to accept the view of Nothnagel and others that they are the result of destruction of the red blood-corpuscles, but would rather attribute them to the direct action of the drug on the tissues, due possibly to the setting free of chlorine, which plays the same *rôle* that, according to Binz, iodine does in the fatty degeneration seen in iodoform poisoning. In confirmation of this may be mentioned the observation of Kast (6), that chloroform on reaching the blood after inhalation sets free its chlorine, which is excreted in the urine. Zeller has found the same to be the case after administration by the mouth.

In a later edition of Casper's work the term "protrahirte Wirkung des Chloroformes" has been substituted for "die chronische Chloroform Vergiftung," originally employed by that author. Ungar very properly objects to this term, as it gives rise to the erroneous impression that death results from the persistent and uninterrupted action of the chloroform, and that the individual remains until death under the persistent influence of the poison in the body. Ungar prefers the term "tödliche Nachwirkung der Chloroforminhalationen," which is certainly better.

Strassmann (7), in 1889, confirmed the observations of Ungar, and showed that chloroform caused an increased excretion of nitrogen in starved animals, thus pointing to the increased destruction of proteids. From these observations he concluded that the fatty change produced by chloroform was a degeneration, and not merely a fatty infiltration. Moreover, he showed that by bleeding the animal before administering the chloroform the fatty degeneration of the liver was more marked. It is im-

portant to note that in two of Strassmann's experiments, in which the animals had been anæsthetised for three and five days respectively, the organs were found to be free of fat sixteen and fourteen days later. Schenk, whose experiments are referred to later, finds distinct evidence of fatty degeneration weeks after an administration lasting only one to one and a half hours.

In the same year (1889) Ostertag (8) experimented on rabbits, guinea-pigs, rats, pigeons, cats and dogs, and was able to confirm the results of Ungar. He sums up his results as follows :—

(1) Fatty infiltration of the liver and fatty metamorphosis of the heart, the skeletal muscles, the kidneys and the stomach, can be produced in various animals as the result of the long-continued inhalation of chloroform.

(2) The fatty metamorphosis of the organs is the result of the action of the chloroform on the blood (destruction of the red blood-corpuscles) and on the cells of the tissues themselves.

(3) Certain individuals show such a strong susceptibility to these actions that they succumb to them at a shorter or longer time after the inhalation.

(4) The fatal after-action of the chloroform is brought about by paralysis of the heart, which may be attended by only slight anatomical evidence of injury to the myocardium, and by a gradual overloading of the blood with carbonic acid.

Ostertag believes that the fatty change in the liver is an infiltration, and that the fat which is deposited there is derived from that which results from the fatty metamorphosis of the heart and kidneys.

In 1890 Thiem and Fischer (9) published under the title "Ueber tödliche Nachwirkung des Chloroformes" the case of a man who died four days after an operation on the knee-joint for the removal of extravasated blood. At the post mortem there was found both fatty infiltration and fatty degeneration of the liver. The kidneys were normal.

In the following year Bastianelli (10) reported three cases in which death occurred from three to ten days after anæsthesia. The chief symptoms were extreme restlessness, severe vomiting, cerebral excitement, followed by collapse and death. Jaundice occurred in one of the cases. The post mortem revealed intense fatty degeneration of the liver, and, to a less extent, of the kidneys and heart. There were also subserous hæmorrhages.

We are indebted to Fraenkel (1892) (11) for the first detailed description of the naked eye and microscopical changes which may result in the human subject from the inhalation of chloroform. Fraenkel made a very careful investigation of the organs in five cases in which chloroform had been administered for from two to four hours. The deaths occurred from forty hours to twenty days after the operations. The heart showed marked fatty degeneration in all the cases. In the kidneys there was marked degeneration of the epithelium, especially of the convoluted tubules, varying in intensity from cloudy swelling to actual necrosis; in one case there was marked fatty degeneration. Although Fraenkel laid less stress on the changes in the liver, he was able, in four out of the five cases examined, to detect a fatty change, especially at the periphery of the lobules. The liver cells were degenerated, and their nuclei often necrosed or badly stained. Fatty degeneration was also observed in the voluntary muscles and in the ascending aorta.

Fraenkel, after comparing the morbid changes above referred to in the parenchymatous organs with those found in patients dying from sepsis, etc., came to the conclusion that the former were specific for the action of chloroform. He lays stress on the fact that he found a considerable deposit of a light orange-yellow pigment in the kidneys and liver, and suggests that its presence seems to lend support to Ostertag's view that the delayed poisonous action of chloroform is due to its destructive action on the red blood-corpuscles. At the close of his paper he refers to the case of a strong muscular man, aged 38 years, who

died sixteen days after resection of the pylorus for carcinoma. In spite of the fact that he was kept under chloroform for four hours, the heart, liver, and voluntary muscles showed no abnormality, and only a very slight parenchymatous degeneration was present in the kidneys. The patient died of gangrene of the lung. He mentions this case because it seems to support the view that idiosyncrasy plays an important part in chloroform poisoning, a point which is brought out also by the experiments of Heintz and others. Fraenkel states that patients no doubt often recover when the morbid changes produced by the chloroform are less severe than those he described. He makes the important observation, however, that the clinical symptoms may be very slight even in cases in which, at the post mortem, the heart, liver, and kidneys are shown to be extensively degenerate.

Guthrie's first paper, entitled "Some Fatal After-Effects of Chloroform in Children," published ten years ago, was the first important contribution to the subject in this country. In this paper he reports nine cases in which death is attributed to the after-effects of the chloroform, and one in which recovery took place. He stated that he was unable to account otherwise for the fatalities after the operations than by supposing (1) that the deaths were due to auto-intoxication; (2) that the fatty condition of the liver, and, therefore, functional disturbance of the organ, existed before the operations; and (3) that chloroform and operation-shock combined aggravated the condition already present, and thus loaded the system with toxic alkaloids which the kidneys, in cases 6 and 7, were unable to eliminate. Continuing, Dr. Guthrie says: "The practical results of such views are—(1) That in no case should chloroform be given to patients suffering from fatty liver; (2) that as it is impossible from physical signs and symptoms to do more than surmise the existence of fatty liver, we must rely on signs of functional inactivity of the liver, as indicated by the excess of alkaloidal substances present in the urine; (3) that the precise nature of such

alkaloidal substances and the best methods of detecting them must be left for further investigation." The conclusions arrived at by Dr. Guthrie do not appear to have been accepted in this country; indeed, no further reference was made to the subject until Dr. Guthrie, in a second paper (12), published in 1903, brought forward further observations in support of his thesis. The title of this paper, viz., "On the Fatal Effects of Chloroform on Children suffering from a Peculiar Condition of Fatty Liver," differs from that of his first paper in order to emphasise his contention that the liver is already fatty before the chloroform is administered, and that the latter merely acts as the "last straw." In all but the first two cases recorded in his first paper, however, there were concomitant factors which probably had as much to do with the fatal results as the chloroform.

Dr. Guthrie states that the four additional cases recorded in his second paper seemed to prove the following: (1) That neither carbolic acid poisoning nor fat embolism will account for these mysterious fatalities; (2) that the severity of the operations has little, if anything, to do with the cause of death; (3) that the only pathological condition commonly found after death is a peculiarly intense degeneration or fatty infiltration of the liver. This condition was found in five of the first series, and in three of the second series of cases . . . ; (4) the only other circumstance common to all the cases was that chloroform had been administered some hours or days before death. We are therefore driven to seek an explanation in the morbid state of the liver which was demonstrated in eight cases out of fourteen, and which can therefore not be regarded as a pure coincidence, and also to inquire again what part (if any) was taken by the chloroform in producing it.

Guthrie states that all authorities seem united in ascribing the pathological changes met with after death to the action of chloroform alone, whereas he formerly held, and still believes, that chloroform only takes the *rôle* of the "last straw" in the matter. Guthrie explains the

deaths as follows: "(1) The liver being, previously to the operations, in an advanced state of fattiness, was probably on the verge of functional inadequacy with regard to elimination of toxins entering the portal circulation, though still able to hold its own in ordinary circumstances. (2) Chloroform, by decreasing oxidation, already deficient, aggravated the condition of fattiness already present, and so lowered the hepatic function that ptomaines or toxins escaped into the general circulation. (3) Chloroform, moreover, by its specific action on the kidneys, prevented the elimination of such poisons by the urine, and was thus, again, immediately concerned in the cause of death." Moreover, it is to be noted that Guthrie holds that in children the form of fatty liver here referred to is altogether peculiar to these cases of fatality following operations under chloroform. Guthrie states that he is familiar with most kinds of fatty liver, but he has never seen the pale fawn or buff-coloured variety, except in such cases as these. The distinction is that the fatty change affects the entire organ uniformly, and is not confined to portions of it. He states that the nearest approach to these appearances is in acute yellow atrophy, but, in this connection he very rightly says that the liver in these cases is not atrophied.

Ambrosius (13) reports a case of pyosalpinx. The operation lasted three hours. Death occurred ninety hours later, with symptoms of gradually increasing collapse. The post mortem revealed no peritonitis. There was fatty degeneration of the renal epithelium, mainly in the collecting tubules, and necrosis of the convoluted tubules. The heart showed fatty degeneration and fragmentation. In discussing the case, Ambrosius calls attention to the persistent rapid breathing, indicating in his opinion some action, not only on the cardiac nerves, but also on the ganglion cells of the central nervous system.

Becker (14) examined a large number of specimens of urine from patients after anæsthesia, and found an increase of acetone in the urine, especially after chloroform. The

duration of the narcosis seemed to be of no importance. Children are apparently more susceptible than adults, and their urine contained a relatively larger amount of acetone. Existing acetonuria, as, for example, in diabetes, was greatly aggravated by chloroform. He holds that acetonuria is due to an increased destruction of albumin. Luzatti largely confirms the observations by Becker. He found, however, that the acetonuria was in proportion to the amount of chloroform inhaled.

Nachod (15) examined the urine in 57 cases of chloroform anæsthesia. The ages of the patients varied from one to fifteen years. He found serum-albumin, nucleo-albumin, and casts, either alone or in various combinations, in a few cases. According to Nachod, existing albuminuria is not increased after chloroform. He describes a parenchymatous degeneration of the kidney in a child dying twenty-four hours after an operation from hare-lip, the condition exactly resembling that described by Fraenkel and Eisendrath (16). Nachod never found glucose in the urine after chloroform, but many have found other reducing substances. He reports, however, a case of glucosuria and diabetic coma occurring after chloroform-anæsthesia in a patient who had sugar in the urine some time previously, but not immediately before the operation. He relates three other cases illustrating the danger of chloroform in diabetics. Referring to the excretion of acetone in the urine of patients after chloroform, he found that acetonuria occurred in 30 out of 57 cases, but seldom lasted more than three days; in 14 cases aceto-acetic acid occurred along with the acetone. Bile pigments were never found, but urobilin was frequently demonstrated, being usually first seen about the second or third day, and continuing till the fourth. Its presence is attributed to the destruction of the red blood-corpuscles. Nachod holds that the changes in the kidneys and the metabolic disturbances leading to the altered urinary excretion are directly due to the action of the chloroform. He concludes by describing a case where death occurred

twenty-four hours after chloroform-anæsthesia, and in which there was fatty degeneration of the convoluted and straight tubules, and degeneration of the nuclei along with healthy glomeruli and no signs of inflammatory mischief; he believes that these are the changes which exist in the cases in which the chloroform has set up albuminuria and the formation of casts. The variations in the behaviour of the kidneys towards chloroform is attributed by Nachod to idiosyncrasy.

Hill Abram (17) examined the urine in 25 cases after anæsthesia, and found acetone present 16 times (64 per cent.). He states that the quantity of the anæsthetic used bears no relation to the degree of acetonuria. Ether, though it always causes acetonuria, does so to a less extent than chloroform. Abram therefore agrees with Becker that acetonuria follows anæsthesia, and that it is increased by it if already present. According to Le Noble the acetone is a product of albumin destruction, but if this be the source the amount of the acetonuria should bear some relation to the amount of the anæsthetic used.

Babaci and Bebi (18) investigated the urine after anæsthesia, 54 of the cases being chloroformed and 41 etherised. They observed albuminuria in 18·89 per cent. of the chloroform cases, and 36·6 per cent. of the ether cases; three of the latter had previous albuminuria, and two died. A third fatal case, in which there was no previous albuminuria, died five days afterwards; the section showed long-standing nephritis. They further studied experimentally the action of chloroform and ether on the kidneys of dogs, rabbits, and guinea-pigs. In the etherised rabbits a diffuse hæmorrhagic nephritis was produced, which tended to pass off spontaneously, leaving a healthy functionating kidney. With chloroform, on the other hand, albuminuria was not so frequently produced, but when present it appeared to be associated with a parenchymatous nephritis of more chronic type.

Steinthal (19) describes what he regards as a typical case of chloroform poisoning after an operation for fistula

of the cæcum, secondary to perityphlitis. The operation lasted two hours. Death occurred five days afterwards. On the second day the patient became jaundiced, the pulse was rapid (130), there was slight albuminuria and considerable hæmaturia, but no casts were present. Death was preceded by coma. The post mortem revealed no peritonitis; there was fatty degeneration of the liver, heart, and kidneys, the latter showing also evidence of commencing hæmorrhagic nephritis.

Ajello (20) examined the urine in 214 patients who had been anæsthetised with chloroform. He found that chloroform produced albuminuria in a varying percentage, as high as 80 per cent. It is usually slight and temporary, however, lasting from two to six days. Casts were seen in 60 per cent. of the cases; they were few and temporary. He considers that chloroform produces considerable disturbances of nutrition, and that it has a destructive action on the blood. The after-effects depend on various factors, viz., the duration of the anæsthesia and the amount of chloroform used, or on some special idiosyncrasy. He says that the results may be very severe where there is previous organic disease of the heart, kidneys, or vessels, or where there is sepsis, diabetes, or cachexia, and, lastly, where there has been repeated anæsthesia at short intervals. Ajello also studied the morbid histology in four autopsies after death from chloroform, and in numerous dogs anæsthetised and killed at various periods. His conclusions are:—

(1) Chloroform produces acute parenchymatous changes of an inflammatory and degenerative character; the degree and intensity of the changes are, to a certain extent, proportional to the duration of the anæsthesia and to the amount of chloroform inhaled.

(2) These changes consist in fatty degeneration and necrosis of the epithelium of the kidneys and liver; and there are also degenerative changes in the heart, striped muscle, spleen, and blood.

(3) These morbid conditions are not definitely character-

istic of chloroform, for they are produced by all toxic substances (generally products of infective organisms) which gain entrance to the internal organs, or which are formed in them.

They generally pass away, leaving the organs perfectly normal; but they can also cause death, especially from uræmia; only seldom do they assume a chronic type.

Chiarleoni (21) has seen three cases of cholæmia after laparotomy, during which the liver was not specially interfered with. He has frequently seen bile pigments in the urine after chloroform anæsthesia, and considers that this is directly due to the chloroform.

Grube (22) has noted the injurious effects of repeated administrations of chloroform on the kidneys. He did not, however, notice any increase of pre-existing albuminuria. He seldom found sugar in the urine after chloroform, but there was almost always an increase in its reducing power. He also noticed increased excretion of uric acid, chlorides, phosphates, and sulphates, indicating, in his opinion, deficient oxygenation as well as destruction of red blood-corpuscles. He also frequently found urobilin, but seldom glucosuria and hæmoglobinuria. He states that the cause of death in such cases is an auto-intoxication.

Zachrissen (23) reports five cases of delayed chloroform poisoning. He also made a careful examination of the urine of 100 cases after chloroform anæsthesia and 94 after chloroform-ether anæsthesia. His conclusions are: (1) In both there is often slight transient albuminuria. (2) In the chloroform cases it appeared in from 24·5 to 38 per cent., while in the chloroform-ether cases it appeared in 21·17 per cent. (3) The frequency appears proportional to the length of the operation. (4) In both casts are often seen, more often in the chloroform cases. (5) If albuminuria or casts be present before the anæsthesia, the condition is generally aggravated. He attributes the above changes to a destructive action on the renal epithelium, and advises subcutaneous injection of saline immediately after the operation.

Eisendrath (16) examined the urine of 139 cases after general anæsthesia, and concludes that—(1) Already-existing albuminuria is aggravated more often by ether than by chloroform. (2) Albuminuria appears more often after chloroform than after ether—after chloroform in 32 per cent., after ether in 25 per cent. (3) The influence of both on waxy kidneys is the same. (4) Cylindruria appears with or without albumin, as frequently after chloroform as after ether, but diminishes quicker after the latter. He describes also the post-mortem appearances in a patient who died from pulmonary embolism eleven days after a herniotomy, chloroform being the anæsthetic used. For the first two days after the operation there was an acute cylindruria, but the urine was always free from albumin. Examination of the kidneys at the post mortem showed cloudy swelling of the epithelium and necrosis of the nuclei of the convoluted tubules, a change which could only have been caused by the action of the chloroform.

It is only reasonable to conclude that if chloroform can produce parenchymatous degeneration in the kidneys, it is almost certain to produce similar changes in the liver and heart, a conclusion which is borne out by post-mortem observations as well as by experiments in animals.

Bandler (24) deals especially with the action of chloroform on the liver. He reports a death after an operation, lasting one hour, for incarcerated hernia. Apart from the hernia, the patient was a strong, healthy man. No antiseptic was used. The symptoms which followed the operation closely resembled those of acute yellow atrophy.

On the second day there was distinct jaundice and the urine contained bile pigments. On the third day the jaundice was very pronounced; the urine contained much albumin and granular hyaline casts. The patient became very apathetic, and passed very little urine; in the evening he became suddenly violent and delirious. Next morning he was completely unconscious, and small hæmorrhages appeared over the abdomen. The urine contained

leucin and tyrosin. Death occurred on the following day. Diagnosis: acute yellow atrophy. Section: wound healthy; no peritonitis; liver intensely yellow, normal in size, and surface smooth; kidney capsules stripped with difficulty, and on section parenchyma pale and bile-stained; heart muscle pale, flabby, with increase of sub-pericardial fat. The trachea and œsophagus showed numerous hæmorrhages in the mucous membranes, and there were others in the skeletal muscles, especially those of the trunk.

Microscopically the liver showed the cells converted into a granular débris mixed with large and small oil droplets, with here and there remains of the nuclei; the outlines of the cells had almost entirely disappeared. The convoluted tubules of the kidneys showed cloudy swelling going on to necrosis. The muscular fibres of the heart were also degenerated.

Bandler regards the case as one of acute yellow atrophy occurring in a previously healthy man after an operation of no great severity. He expresses the opinion that the condition resulted from the action of the chloroform on a liver previously fatty, and draws attention to the investigations of Pohl (25), who holds that the chloroform which combines loosely with the red blood-corpuscles is set free in the presence of cholesterin and lecithin, and that the degenerative changes in the kidneys are due to the direct action of the chloroform, or are secondary to the liver condition. While Bandler states that this is the first published case of acute yellow atrophy due to chloroform, he is of opinion that some of the recorded cases of this disease which have followed operation were really due to chloroform. He mentions a similar case of von Erlach's in which the patient, before operation, suffered from gastric catarrh and simple catarrhal jaundice.

Bandler then gives the results of his experiments on the action of chloroform on the liver of rabbits and dogs. Comparative investigations made with ether: Out of the four dogs and four rabbits anæsthetised with ether, Bandler only found a very slight degree of fatty change

in the liver of one of the dogs, thus confirming the observations of Strassmann.

Marthen (26) reports a fatal case of delayed chloroform poisoning in a woman, aged 34, who was anæsthetised for forty minutes for the removal of a large number of teeth. She died on the fourth day. The symptoms were: persistent vomiting, increasing jaundice, restlessness and delirium, followed by lethargy, Cheyne-Stokes breathing, and, finally, heart failure. The urine contained albumin and hyaline and fatty casts.

Post-mortem.—General icterus; no cutaneous hæmorrhages; heart flabby, yellowish brown, and streaked with sub-endocardial hæmorrhages; kidneys bile-stained, enlarged, flabby; cortex thickened, and a few hæmorrhages in the pelvis. Liver smaller than normal, yellow, very flabby, *surface smooth;* on section, lobules unusually distinct, centres dark red, peripheries intensely yellow, due to fatty change.

Microscopic examination.—Kidneys: cloudy swelling and fatty degeneration, especially of the convoluted tubules; glomeruli normal; golden yellow pigment in the epithelium and between the tubules.

Heart: cloudy swelling with some increase of pigment; striation for the most part retained; a few fibres homogeneous; many of the fibres transversely segmented; no fatty degeneration.

Liver: cloudy swelling; fatty infiltration at the periphery of the lobules; nuclei seen imperfectly at the peripheral and not at all in the central zone; some diffuse thickening of the connective tissue, not only in the portal tracts, but also in the interior of the lobules; fatty change best seen in the peripheral zones; a small amount of golden yellow pigment in the peripheral zones.

In reviewing the case from a clinical point of view, Marthen emphasises the jaundice, the condition of the urine, and persistent vomiting; the latter symptom, he says, should always call for a careful examination of the urine. Along with Fraenkel, he points out that the

morbid condition of the urine is by no means a measure of the extent of the parenchymatous degeneration in the kidneys.

Heintz (27) records the clinical history and the results of the post-mortem examinations in eight cases in which death was attributed to delayed chloroform poisoning. He performed a series of experiments on rabbits in order to ascertain the pathological changes produced by chloroform a relatively short time after one, two, or three inhalations, each lasting two hours. Control observations were made on rabbits to which no chloroform had been administered. In one case the animal was anæsthetised six times at intervals of two or three days for the purpose of ascertaining the maximum effect of the chloroform. The animal died during the sixth administration. It was found that there was a considerable difference in the susceptibility to the action of chloroform in the different animals. Various combinations and various degrees of degeneration occurred in the heart. These changes consisted in loss of transverse striation, fibrillation, and segmentation of the muscular fibres, along with disappearance of the nuclei. Unlike previous observers, he could not detect any evidence of fatty degeneration of the fibres. The changes in the liver, however, were the same as those produced by other experimenters, and consisted especially in fatty degeneration. As in the human subject, the kidneys were less affected than the heart and liver. From the post-mortem appearances found in the human subject, and the experimental results obtained in rabbits, Heintz comes to the conclusion that a single prolonged inhalation of chloroform is able to give rise to subsequent pathological changes in vital organs which may be fatal in twenty-four hours or several days, and this in spite of the fact that the patient had completely come out of the chloroform.

Heintz has shown that the changes in the internal organs are by no means proportionate either to the amount of he anæsthesia or to the amount of chloroform employed; he concludes, therefore, that idiosyncrasy plays an im-

portant part. The changes in the heart were found to be practically the same in the rabbits as those revealed at the post mortems in the human subject, except that in the former the nuclei were not necrosed. The changes in the liver and kidneys were practically the same in both.

Heintz agrees with Ungar in attributing the degenerative changes in the organs to the direct toxic action of the chloroform, and he regards the fatty change as a degeneration rather than an infiltration. He is of the opinion that the changes in the kidneys are so slight as compared with those found in the other organs that death cannot be attributed to uræmia.

Heintz draws the following conclusions from his observations: (1) That the occurrence of delayed chloroform poisoning must be admitted. (2) That death may take place five days or longer after the administration. (3) It may follow either a single prolonged administration or repeated administrations at short intervals. (4) Characteristic changes are found in the organs of human subjects dying from prolonged chloroform poisoning. (5) The changes consist in degenerations of the cells, the result of the direct toxic action of the chloroform. (6) The changes in the heart consist in degeneration of the muscular fibres and disappearance of the nuclei. (7) The liver shows fatty degeneration and necrosis of the epithelium. (8) The kidneys, which are not so rapidly affected as the heart and liver, show cloudy swelling, and, in severe cases, necrosis of the epithelium. (9) The chief clinical symptoms are: severe and long-continued vomiting, rapid pulse and physical disturbances. (10) Slight degenerative changes in the organs may be recovered from. (11) Death usually takes place suddenly, with symptoms of collapse. (12) The cause of death cannot be attributed to functional disturbances of a single organ. (13) Death is the result of a general intoxication which produces pathological changes in most of the vital organs.

Schenk (28) made investigations on apes and dogs with

the object of ascertaining how soon the fat appears in the organs after the anæsthesia, and when the organs become free from fat again. In healthy dogs in which no chloroform was administered he found a certain amount of fat in the heart, kidneys, and liver, in the latter in the form of the finest droplets within the liver cells. In each experiment, before giving the chloroform, a small piece of liver was excised for purposes of comparison. The animals were killed at various periods, viz. immediately after anæsthesia, and at the end respectively of 12, 24, 48 hours, and several days, up to four weeks. He performed two experiments with ether for purposes of comparison, and found that, while fat appeared in the liver, it was very slight in comparison with what was found after chloroform. He deals in detail only with the changes produced in the liver, because it was only in this organ that a comparison was made with the condition before anæsthesia. His observations on the heart and kidneys confirm those of previous investigators. The experiments show that the fat which appears in the liver some hours after chloroform anæsthesia in some instances does not disappear for days, indeed sometimes not for weeks. He was unable to confirm Strassmann's observations that the fat disappeared from the liver fourteen days after somewhat prolonged administrations: in all his experiments fat could be detected at this period in spite of the fact that the anæsthesia was not so prolonged as in Strassmann's experiments. He concludes by cautioning gynæcologists against giving chloroform a few days after its employment for diagnostic purposes.

Salen and Wallace (29) report two cases. The first, which resembles Bandler's cases, was that of a woman, aged 20 years, who had undergone a laparotomy of two hours' duration for salpingo-oophoritis. Jaundice appeared the day after the operation, the other symptoms being great weakness, rapid and feeble pulse, collapse, and death on the third day.

Post-mortem.—Wound and peritoneum healthy; no

swelling of spleen; advanced fatty disintegration of liver; fatty degeneration of heart and kidneys.

In the second case death occurred two days after operation for appendicitis. The urine contained albumin and casts, and complete anuria followed.

Post mortem.—Wound and peritoneum healthy; spleen normal; marked fatty degeneration of heart, liver and kidneys.

Dorner (30) publishes the case of a child, aged $3\frac{1}{2}$ years, who died forty-two hours after Bassini's operation for hernia. The operation was followed by slight albuminuria, a gradual rise of temperature, and increasing cardiac weakness. The wound was healthy. At the post mortem there was found an enlarged thymus along with hyperplasia of the lymphatic system. The internal organs showed the characteristic fatty degeneration seen in cases of delayed chloroform poisoning. In this case the *status lymphaticus* no doubt rendered the organs specially susceptible to the toxic action of the chloroform.

Poroschin (31) gives the results of thirty experiments on the changes in the organs of dogs after chloroform anæsthesia. In addition to the degenerations in the various organs described by previous observers, he found degenerative changes also in the cardiac ganglia, and in the respiratory centre, also hyaline degeneration of the small arteries of the heart, stomach, and Malpighian bodies of the spleen.

Cohn (32) reports the case of a robust woman, aged 21 years, who was operated on for double salpingo-oophoritis. The operation lasted one hour, and the patient took a large quantity of chloroform. She remained in a half-conscious condition the whole day. During the night she was very restless, and had to be tied down in bed. She was, however, able to answer questions quite intelligently; pulse and temperature normal. On the following day she became drowsy and apathetic. The next day there was slight jaundice, and in the evening she again became excited and screamed out; temperature normal. Next

day she became comatose, cyanotic, and distinctly jaundiced. A few cubic centimetres of turbid urine were withdrawn, and found to contain albumin, granular and epithelial casts, a few blood-corpuscles, and some bile pigments; no sugar. She died at 8.30 p.m., five days after operation.

Post-mortem.—No cause of death connected with the operation. Liver yellow, flabby, not enlarged. Microscopical examination showed a fatty change in the peripheral and outer intermediate zones, the nuclei being obscured by the fat globules; in the centre of the lobules an abundant deposit of yellow pigment; no evidence of an old inflammatory lesion. The heart showed foci of fatty degeneration along with some pigment. The kidneys showed marked cloudy swelling of the epithelium of the convoluted tubules; glomeruli normal.

The author states that death was due neither to sepsis nor to antiseptic poisoning, and that the only satisfactory explanation of the symptoms and post-mortem appearances was that the chloroform set up an acute intoxication.

Ballin (33) reports what he believes to be a recovery from acute yellow atrophy following appendicectomy. The symptoms were certainly suggestive of acute yellow atrophy. Wersung is mentioned as having collected sixteen recoveries from the disease. Mintz, on the other hand, regards post-operative cases of acute yellow atrophy as essentially septic, because in a case very similar to Ballin's he found bacterium coli in the liver, and a thrombotic process in the duodenal artery. Ballin points out the significant fact that no case of acute yellow atrophy has been recorded after the administration of ether.

Waldvogel (34) states that the original idea that acetone arises by the oxidation of carbohydrates and that it is also a product of the destruction of proteids is untenable. Recent investigations have demonstrated that fat is the true source of acetone, as the latter is increased during starvation, and in diabetes. Carbohydrate, on the other hand, causes the acetonuria produced during

starvation at once to disappear. Waldvogel points out that it is of interest to the surgeon to know that acetonuria after anæsthesia is due to fat destruction caused mainly by the toxic action of the anæsthetic.

Rydygier (35) in a paper dealing with kryoscopic observations on the urine after chloroform anæsthesia, states that chloroform does not set up any renal insufficiency when the kidneys are normal, but that it has a serious effect when the kidneys are diseased. The presence of nephritis he regards therefore as a contra-indication to its use.

Foerster (36) records two cases (from the clinic of Professor Schede, of Bonn) which are "with certainty to be regarded as deaths due to the after-action of chloroform." The first is that of a girl, aged 4 years, who died forty-eight hours after an osteotomy of both femora. The duration of the anæsthesia was fifty minutes. The operation was followed by increasing cardiac weakness and persistent vomiting, the vomited matter being almost black in colour.

Post-mortem—Liver.—Somewhat enlarged. Surface smooth and of an orange-yellow colour; on section the whole organ was of the same uniform light-yellow colour, and the individual lobules could not be recognised. Sections stained with sudan showed an extreme fatty change, each lobule being affected throughout, but more so in the peripheral zones. The outlines of the cells were irregular and often quite obscured, but the nuclei had not disappeared.

Kidneys.—Cortex of a dirty yellow colour, and somewhat anæmic; medulla dark red. Microscopically (sudan staining) the glomeruli were everywhere healthy and their nuclei well stained. The convoluted tubules were saturated with the stain, and to a less extent the looped and collecting tubules.

The heart showed a patchy fatty degeneration.

The second case was that of a girl, aged 11 years, who died on the fifth day after operation for a localised abscess, the result of appendicitis. Previous to the operation there

was no evidence of any disease of the internal organs. For the first three days after operation the patient's condition was in every way satisfactory. During the fourth night she was very restless, and vomited frequently. Next day there was slight jaundice, she became somewhat delirious, and cried out constantly. The following day there was persistent coffee-ground vomiting, the pulse and temperature were elevated, and she died comatose. The urine contained bile pigments, but no albumin.

Post-mortem.—No suppuration or deposit of lymph beyond the immediate site of the abscess.

Liver.—Somewhat congested; deeply jaundiced; lobules very distinct, centres congested, peripheral zones yellow and swollen. Sections stained with sudan showed the same fatty change as in first case, the fat droplets being smaller and fewer in the central zones.

Kidneys.—Cortex thickened, yellow and cloudy. Microscopical examination revealed similar changes to those met with in the first case, except that only a few of the straight tubules contained fat.

Heart.—Muscular substance pale and cloudy, with a few punctiform hæmorrhages under the epicardium. The microscope showed, in osmic acid preparations, a finely granular fatty condition, uniformly distributed through the whole muscular substance of the heart.

Foerster points out that neither child had suffered from any constitutional disease, and that as far as he could ascertain there was no organic disease of their internal organs.

He asserts that the morbid changes found, more especially in the kidneys, afford sufficient proof that death was due to the after-action of the chloroform.

We shall now pass on to the consideration of our own observations; but before doing so we desire to express our thanks to Mr. C. B. Paul, F.R.C.S.Ed., for the assistance he has given us in preparing abstracts of the literature.

In a paper on "The Radical Cure of Hernia in Infants

and Young Children," read at the annual meeting of the British Medical Association, Cheltenham, 1901, one of us had to report three deaths in his first 100 cases, one of which he considered to be a typical case of delayed chloroform poisoning. It was that of a healthy girl, aged 4½ years, operated on on August the 1st, 1900, for a left reducible inguinal hernia. The operation consisted in the ligature and removal of the sac, and the closure of the canal by two catgut sutures. No drainage; collodion dressing. The anæsthesia lasted twenty minutes. On the evening of the operation the pulse was a little quickened, and the temperature 101° F. The patient passed rather a restless night and vomited frequently. Next morning (August the 2nd) there was a slight icteric tinge of the skin. The vomiting continued at intervals all day. In the evening the patient looked rather collapsed; pulse 110, but quite regular; temperature 100° F.; tongue moist and clean.

August the 3rd.—"Has not kept anything in stomach since operation; jaundice quite distinct; urine contains a considerable quantity of albumin."

Evening.—"Looks decidedly ill; pulse 140; temperature 103° F. Has only passed 6 ounces of urine in the last 24 hours. Bowels have not acted."

August the 4th.—"Very ill this morning; pulse 156; has been delirious for the last six hours; no urine secreted since 4 p.m. yesterday. Died comatose at 6.30 p.m. The temperature shortly before death reached 104·8° F."

Post mortem.—Wound and peritoneum healthy. Liver very yellow to the naked eye; intensely fatty. Microscopically the liver cells were filled with oil globules of various sizes and their outlines obscured; in some places the cells appeared to be disintegrated. In the kidneys there was marked cloudy swelling, almost amounting to necrosis of the epithelium, especially of the convoluted tubules; the nuclei stained either imperfectly or not at all. The sections were not stained specially for fat.

As some surgeons do not seem to have recognised the

condition, it may be as well, in the first place, to describe somewhat more in detail the clinical and pathological features of a case which occurred subsequently to the above, and which we believe to be typical of the condition.

Jane R—, aged 8 years, a well-nourished, somewhat florid and healthy-looking girl, with no evidence of organic disease, was operated on for genu valgum in the Royal Hospital for Sick Children, at 11 a.m., on October the 21st, 1902. As the abnormality was due to a well-marked overgrowth of the inner part of the lower end of the diaphysis of the femur, a wedge of bone was removed from this region by a modification of the method originally advocated by Chiene. There was no difficulty about the operation, and no shock. The total duration of the anæsthesia, including induction, was twenty-two minutes. The patient was a little sick after her return to consciousness. There was no further vomiting during the whole of the afternoon, and the child's condition was in every respect satisfactory. Later in the evening vomiting again set in, and continued at intervals throughout the night. The vomited matter, which consisted at first of gastric juice mixed with a little bile, became more copious and of a dark green colour, and finally presented the characteristic beef-tea-like appearance described by Guthrie. She complained of great thirst, cried out for drinks, and became very restless. Every now and then she attempted to throw off the clothes, and would frequently clutch hold of the bars of the crib and pull herself into awkward positions. In the intervals between these excited periods she appeared to be rather listless, and did not complain of any pain. The pulse became increasingly rapid and feeble. The breathing was natural. There was no involuntary passage of urine or fæces. The following morning the vomiting ceased, and the patient's condition was considered by Dr. Price, the house-surgeon, to be more satisfactory. She was, however, a little

flushed, and the pulse was still small and quick. At 1 p.m. the probationer tidied her bed, and gave her the drink she asked for. An hour later the sister noticed the child was looking collapsed, grey, and slightly cyanosed. The pulse was almost uncountable, unconsciousness rapidly supervened, and the patient died suddenly at 2 p.m.—that is, twenty-six hours after the operation.

Post-mortem, October the 23rd.—The body was that of a well-developed child. Rigor mortis was general. Post-mortem lividity distinct. There was a recent osteotomy wound over the lower end of right femur. On opening up the wound the parts looked perfectly healthy; there was diffuse hæmorrhagic infiltration of the tissues for some distance around, but no evidence of septic infection.

Thorax.—No excessive exudation into either the pericardial or pleural sacs. The thymus was distinctly enlarged, extending down over the surface of the pericardium.

Heart.—Pericardium showed nothing abnormal. Right side of the heart contained dark fluid blood and soft post-mortem thrombus; the right side of the heart was not specially distended; the left ventricle was firmly contracted; the aortic and pulmonary valves were competent, and the cusps at the various orifices showed nothing abnormal; the myocardium itself was rather pale, but quite firm; there was no mottling or other distinct naked-eye evidence of fatty degeneration.

The right lung.—Scattered over the visceral pleura, especially towards the root of the lung, were numerous small subpleural hæmorrhages, the largest measuring about $\frac{1}{8}$th inch to $\frac{1}{4}$th inch in diameter. There were also numerous areas of lobular collapse. Neither the root of the lung nor the large branches of the pulmonary vessels showed anything abnormal. The large bronchi were congested, and contained frothy mucus, at places blood-stained. On section the lung as a whole was congested, and there were numerous fairly sharply outlined areas of hæmorrhagic infiltration, giving a distinct mottled appear-

ance. There was no definite pneumonic consolidation at any part. There were no distinct pyæmic foci, and no tuberculosis. The left lung showed practically the same condition, but the hæmorrhagic areas were rather smaller than on the other side and not so numerous.

Abdomen.—No peritonitis or other abnormality in the peritoneal sac. The stomach and pancreas showed nothing abnormal. Intestines presented no abnormality beyond some prominence of the lymphatic tissue towards the lower end of the small intestines. The mesenteric glands were slightly enlarged, soft, and pale, and showed no evidence of tuberculosis.

Liver.—Distinctly enlarged; surface smooth, and the colour intensely yellow, almost a canary tint. Scattered on the surface were some small dark red spots and streaks representing distended vessels; there were, however, no distinct hæmorrhages. On section there was seen to be an intense degree of fatty change uniformly distributed through the whole organ. The condition evidently affected the whole of the hepatic lobule, but at places the peripheries of the lobules had a more intensely yellow tint. There was no evidence of an infiltration of any kind in the portal tracts, and there was no indication to the naked eye of septic foci. Gall-bladder and suprarenals normal.

Kidneys.—Both were slightly enlarged and rather soft in consistence. The capsules stripped readily, leaving a smooth, extremely pale surface. The stellate veins were not unduly prominent. On section the most striking feature was the intense pallor of the cortex, both superficial and deep. The cortex was also slightly swollen. In contrast to the pale cortex, the pyramids were rather prominent, but were not in reality unduly congested. The naked-eye appearance suggested an intense degree of cloudy swelling rather than diffuse fatty change. There was no yellow tint in the pallor. The Malpighian bodies were not apparent; the vessels showed nothing abnormal; there was no hæmorrhage either in the cortex or in the

pelvis; there were no septic foci to be seen. Bladder showed nothing abnormal. The spleen was not enlarged and was fairly firm. On section there was slight pallor. The Malpighian bodies were not unduly prominent.

The brain showed nothing abnormal to the naked eye.

Bacteriological.—Cultivations were made on agar and blood-agar from the hæmorrhagic areas in the lungs. In twenty-four hours numerous small clear transparent colonies had developed.

On microscopical examination the growth proved to be one of pneumococcus, giving all the staining properties of that organism. Films made directly from the hæmorrhagic areas in the lungs were specially examined for the presence of bacteria and fat. No bacteria were found, but some very minute globules of fat could be demonstrated both free and inside endothelial cells.

Histology of the lesions.—Pieces of the various organs were fixed and hardened in alcohol, and also in 10 per cent. formalin. These latter were specially examined for fat, sudan, and $\frac{1}{6}$th per cent. osmic acid being the stains used. The tissues were also examined for bacteria. Sections from the tissues in the neighbourhood of the wound showed diffuse hæmorrhagic infiltration of the tissues, with a few polymorphonuclear leucocytes among the effused red blood-corpuscles. There was no histological evidence of a local septic infection, and no bacteria could be demonstrated.

Heart.—In many places the muscle-fibres showed no abnormality. No fat could be demonstrated; here and there the fibres were somewhat granular and the transverse striation not quite distinct. There was no transverse segmentation of the fibres, and the nuclei everywhere stained perfectly; there was no pigmentary deposit and no special hyperæmia.

Lungs.—Sections stained with hæmatin and eosin showed areas of hæmorrhagic infiltration into the alveoli. Here and there a polymorphonuclear leucocyte could be seen, and there was some desquamation of the endothelial

DELAYED CHLOROFORM POISONING.

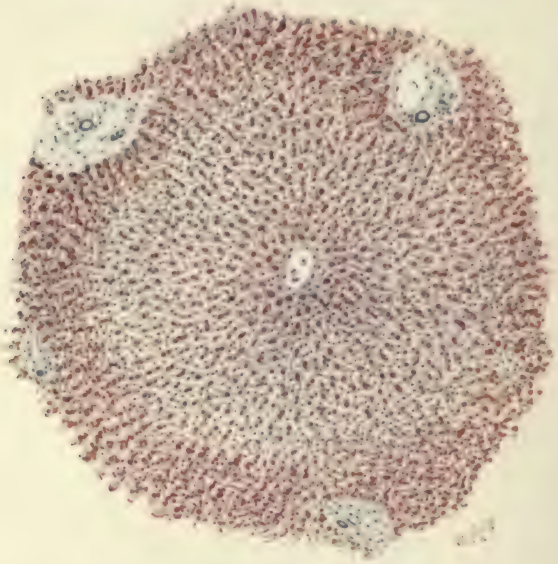

Fig. 1. Hæmatin and Sudan 3. ×80.

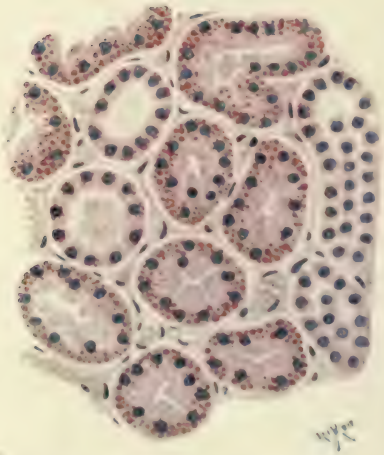

Fig. 2. Hæmatin and Sudan 3. ×250.

To Illustrate Mr. Stiles and Dr. Stuart M'Donald's Article.

Liver and Kidney from patient Jane R.

lining. The alveolar capillaries were congested; the walls of the bronchi were also unduly hyperæmic, but there was no distinct evidence of an inflammatory change in either the bronchial mucosa or in the lung alveoli.

Sections stained by Gramm's method showed a few bacteria: some were morphologically identical with pneumococci, others were longer bacilli, not resembling any particular pathogenic form, and were thought to be probably putrefactive in nature. No bacteria were demonstrated in the lung alveoli. A large number of sections from different parts of both lungs were specially examined for the presence of fat. A very few capillaries in the alveolar walls were found to be blocked by short columns of oil giving the characteristic staining reaction with osmic acid and sudan. Very minute droplets were also demonstrated free in the alveoli, in the endothelial cells lining the alveoli, and in some of the desquamated endothelial cells. At places, too, there was an appearance of similar minute droplets in the endothelial cells of the alveolar capillaries; some very minute droplets free and inside desquamated cells were also seen inside some of the bronchi.

Liver.—Paraffin sections stained by hæmatin and eosin showed some fibrous thickening of Glisson's capsule, not amounting, however, to an actual cirrhotic condition. There was no cellular infiltration in the portal tracts. The liver cells could not be satisfactorily demonstrated owing to the intense vacuolation, especially at the periphery of the lobules, the vacuoles representing spaces from which fat had been dissolved. There was evident degeneration of the cells, especially in the central zones, but the nuclei for the most part stained perfectly well. The most successful preparations were obtained by an ordinary freezing method, and stained by sudan, counter-stained with hæmatin. In those sections (Plate I, fig. 1) an intense fatty change in the cells was well demonstrated. In the central zones of the lobules the cells appeared rather diminished in size, the capillaries between the

columns of cells being correspondingly dilated. The cells contained a great number of minute droplets of oil, which for the most part did not tend to coalesce. The nuclei were frequently obscured by the amount of oil present, but where seen were stained perfectly well. Immediately around the central venule many of the cells appeared to be completely disorganised. This change in the central zone extended into the intermediate zone, but the cells in the peripheral zones presented rather a different appearance. Here, for the most part, the oil droplets had coalesced into large globules, which displaced the nuclei to the sides of the cells. Where seen, however, the nuclei were stained perfectly well. There was no part of the hepatic lobule free from fatty change. The walls of the sub-lobular and hepatic veins were somewhat thickened, and in some of them, particularly in some of the hepatic veins, quite a considerable amount of oil could be demonstrated. Much of this appeared as free droplets. At places the droplets were grouped as if resulting from disintegration of a cell, and one or two distinct liver cells were seen closely resembling the disintegrated cells lying around the central hepatic venule. No fat was demonstrated in branches of the hepatic artery.

Kidneys.—Sections prepared and stained as above showed an intense degree of degenerative and fatty change in the renal parenchyma (Plate I, fig. 2). The change was practically universally distributed throughout the organ, the convoluted tubules, loop tubules, and collecting tubules all suffering. There was distinct cloudy swelling, with a tendency to separation of the cells from one another in the convoluted tubules. The fat was present as minute droplets, not tending to coalesce, and especially situated between the nucleus and base of the cell. The nuclei throughout stained almost perfectly. There was no pigmentary change in the cells; there was a certain amount of granular precipitate in the tubules and between the capillary tuft and Bowman's capsule, but the Malpighian bodies themselves showed nothing abnormal. In none of

the renal vessels was any oil demonstrated. There was no leucocytic emigration at any point, and no cellular proliferation could be made out. Sections of the suprarenals, pancreas, and brain showed nothing abnormal. In none of them could fat be demonstrated in the vessels.

Spleen.—Well-marked hyaline degeneration in some of the splenic arterioles and some proliferation of the endothelial lining of pulp sinuses.

The cause of death in such cases has been variously attributed to chemical poisoning from antiseptics, to fat embolism, to sepsis, and lastly, to delayed chloroform poisoning. We shall now consider these *seriatim*.

1. *Antiseptics.*

As carbolic acid was the only antiseptic employed in this case, we need not consider the toxic action of other antiseptics. That it is necessary, however, to consider the part played by carbolic acid is evident from the criticism offered to Guthrie's paper. In children it is well known that death may result from the absorption of carbolic acid by the skin. One of us who had the misfortune to witness a fatal case which occurred in the Edinburgh Royal Infirmary in 1887 advised Dr. Kruger, the house-surgeon in charge of the patient, to take up the subject of the absorption of carbolic acid by the skin for his M.D. thesis. Dr. Kruger's observations, as far as they refer to children, were made in the wards under the care of one of us at the Royal Hospital for Sick Children. The thesis, for which Dr. Kruger was awarded the Syme Surgical Fellowship, has, I regret to say, not been published. I may, however, refer to the more important results which Dr. Kruger obtained.

Before doing so, however, let me refer shortly to the case which led to the investigations. It was that of an exceptionally strong and healthy male infant, aged 3 months, the subject of double talipes equino-varus. At 1 p.m. on the day previous to that fixed for the operation

the lower limbs, after being washed with soap and hot water, and rubbed with ether, were poulticed with bicarbonate of soda for four hours. A 1 in 60 carbolic poultice (extending up to just below the knees) was then applied for four hours. A weak boracic acid poultice was applied overnight. At 9.30 a.m. the following morning the limbs were again washed up, and a 1 in 60 carbolic poultice reapplied. Owing to the admission of an urgent case, the operation on the child had to be postponed. At 1.30 p.m., four hours after the application of the second carbolic poultice, the child became lethargic, and uttered "peculiar choky cries." At 8 p.m. he was pale, semi-comatose, and perspiring freely; pulse small, and very rapid; breathing shallow and frequent; pupils contracted; eyeballs staring and turned up. There was slight spasmodic twitchings of the upper extremity, and the temperature had risen to 102° F. At 9 p.m. the pulse was scarcely perceptible, and there was persistent vomiting. The urine was almost black in colour, and gave after distillation a strong reaction of carbolic acid, with bromine water and ferric chloride. The child died comatose at 5 p.m. the following morning, 15½ hours after onset of the symptoms.

At the sectio the liver showed well-marked furrows and grooves not unlike the appearance of a syphilitic liver, but on section the organ appeared to be quite normal. Unfortunately, no mention is made of its microscopic appearances. The kidneys gave absolutely no evidence to the naked eye of any catarrhal or inflammatory change. The microscope, however, showed well-marked cloudy swelling and some necrosis of the cells of the tubules. The vessels were markedly congested, and there were small hæmorrhages into the tubules.

Kruger has shown that when a 1 in 40 carbolic poultice is applied to the skin of an adult, 26·5 per cent. of the carbolic acid which enters the system is absorbed in the first two hours, and half that quantity in the next two hours. Relatively more carbolic acid is absorbed by increasing the skin area to which it is applied than by increasing

duration of its application. Moreover, it was shown that if the skin be previously shaved and scraped with soap and water, more than double the amount of phenol is absorbed within periods of two and four hours respectively than when the skin has had no previous cleansing.

In the case of children, 41·3 per cent. of the total amount of phenol which enters the system is absorbed in the first two hours; in other words, children absorb about 75 per cent. more than adults do during the first two hours. This fact, combined with the greater vulnerability of the child's nervous system, is quite enough to account for the number of cases of carbolic intoxication which have been recorded as having occurred in children. Dr. Kruger, in an investigation made on one of us, found that as a result of purifying the hands and forearms with a mixture of green soap, spirit and lysol, followed by 1 in 20 carbolic acid, and of dipping the hands frequently in 1 in 60 carbolic during the operation (occupying together 2½ hours), 355·35 mgrs. of carbolic acid were excreted in the urine during the subsequent 28 hours—the quantity corresponding roughly with what is absorbed when a 1 in 40 carbolic poultice is applied over 72 square inches of skin surface for four hours.

The only signs of injurious action observed in the subjects of Dr. Kruger's experiments were carboluria in several instances, and in one case considerable restlessness during the night of the application of the phenol.

In the cases reported by Clement Lucas and Arbuthnot Lane in which carbolic acid coma was induced by the application of carbolic compresses to the skin in children, the chief symptoms were collapse, weak-running pulse, and quick, shallow respiration, sweating, repeated vomiting, and lethargy, passing into coma. In one of the three cases there was also screaming, and this patient "vomited some dark fluid which appeared to be imperfectly digested beef-tea." It is evident, therefore, that, apart from the carboluria, the symptoms of a toxic dose of carbolic acid absorbed by the skin bear a close resemblance to those

which are supposed to be due to delayed chloroform poisoning. Moreover, as in the latter, there is the same interval of well-being between the application of the phenol and the onset of the symptoms. The rapidity with which carbolic acid is absorbed by the skin, and the fact that several hours may elapse before any toxic symptoms manifest themselves, render it important that the surgeon should inquire into the possibility of carbolic poisoning before attributing symptoms after operation, such as we have mentioned above, either to delayed chloroform poisoning or to some condition associated with the operation itself, or to wound infection.

Since Dr. Kruger made these observations, the application in the Edinburgh Children's Hospital of carbolic acid in the form of a poultice dressing has been discontinued. In the case we are considering carbolic acid, in the form of a 5 per cent. solution, was one of the substances used to purify the skin the night before the operation; but as it was immediately washed off with sterile water, and an antiseptic dressing applied, the amount of the drug absorbed must have been so slight that carbolic acid poisoning may be definitely excluded.

2. *Fat Embolism.*

The possibility of fat embolism as a cause of death in such cases has suggested itself to many. Watson Cheyne, in criticising Dr. Guthrie's original paper, laid stress on this as the probable cause of death in some of the cases, and considered that the absence of a careful examination of the lungs and brain was an unfortunate omission. One reason why the possibility of fat embolism suggested itself in our case was that, during the operation, which consisted, as already stated, in the subperiosteal removal of a wedge of bone from the inner aspect of the lower end of the diaphysis of the femur, about a teaspoonful of yellow fluid fat was seen to exude from the bone. It should be stated, however, that one of us, who is in the

habit of performing this operation for knock-knee, has since observed this on several occasions without any untoward result. The circumstances, too, that death occurred quite unexpectedly, and was preceded by some cyanosis, seemed to add to the possibility of fat embolism. A careful search was therefore made for evidence in favour of this view.

It must be admitted that many of the symptoms which were present in our case, and which Guthrie regards as typical of delayed chloroform poisoning, may be seen in severe cases of fat embolism. It is now generally admitted that fat embolism is far from uncommon, and that in mild degrees it is unattended with symptoms. In the fatal cases, which, on the other hand, are very rare, no difficulty is found in demonstrating microscopically a large amount of fat, at any rate in the lungs. Welsh states that considerably over one half of the pulmonary capillaries may be blocked by cylinders and drops of oil. In our case fat was present in small amount, many sections from different parts of the lungs being examined before blocked capillaries were found, and though it is stated that in such cases fat in the systemic capillaries may be only slight, in the present case it could not be demonstrated in a single vessel on the aortic side of the circulation.

With the presence of multiple hæmorrhages in the lungs, along with a small amount of fat in a few of the capillaries, it might at first sight appear that we need not go further for an explanation of the death. In order to ascertain the extent of the blocking a large number of sections were made of different parts of both lungs. It is to be noted, however, that the actual emboli were very few in number, and were only seen in minute vessels.

Next, as to the possible source of this fat. The most natural explanation, of course, would be that it came from the bone. Although this cannot be definitely disproved, we are of opinion that there is strong evidence pointing to the liver as the source. As already noted in the description of the morbid histology of the liver, the

disintegration of the cells extended to the central zones, and the enormous accumulation of fat in them, coupled with the presence of a considerable amount of oil in some of the large hepatic veins, seems to us to account for all the fat present in the lungs. Even admitting the derivation of some of the fat from the bone, we are strongly of opinion that, considering its small amount, its presence must be regarded merely as an incident in the case, and that the fatal termination was not to be attributed to it. We are quite at one with Dr. Guthrie in the view that fat embolism is not such a potent cause of death as many believe. It is a remarkable fact that few cases of death from undoubted fat embolism have been recorded in recent years, and we are inclined to believe that many cases which were so recorded in the pre-antiseptic days were in reality cases of septic phlebitis with subsequent pulmonary infarction.

3. *Sepsis.*

As long ago as 1874 Koch, in an article on "Chloroform and its Employment in Surgery," states that, in his opinion, a glance at the literature suffices to show that death in cases of so-called chronic chloroform poisoning is not due to, or hastened by, the chloroform, but is the result of intense septic poisoning. Many recent writers, on the other hand, hold that some deaths which have been attributed to delayed shock or acute sepsis are in reality cases of delayed chloroform poisoning. It must be admitted at once that there is often great difficulty in distinguishing the two conditions on clinical grounds alone, because the symptoms described in connection with delayed chloroform poisoning, viz., severe and persistent vomiting, small and rapid pulse, albuminuria, and occasionally jaundice, restlessness, cerebral excitement, and coma, may be met with also in cases of acute sepsis—for example, in fulminating appendicitis. In our present case, as far as can be judged from the naked-eye appearances, the wound

was absolutely healthy. Films from the blood-stained serum in its vicinity showed no great increase of polymorpho-nuclear leucocytes. Further, microscopic sections of the edges of the wound gave no histological or bacteriological evidence of a local infection. The multiple hæmorrhages in the lungs made us suspicious of a septicæmic condition. As has already been mentioned, cultures from the lungs yielded a pure growth of the pneumococcus It is well known, however, that the pneumococcus may be cultivated from the respiratory mucosa without a pneumococcal infection being necessarily present, and it is interesting to note that in the sections of the lungs stained for bacteria none were found except in the bronchi, and that along with the few pneumococci other bacteria, probably putrefactive, were present.

Histologically, too, there was no evidence of pneumonia. The fact that pneumococci were present is interesting, however, as it is known that fat embolism is associated with a pneumonic condition, and quite possibly the vascular disturbance paves the way for infection by pneumococci accidentally present in the upper respiratory vessels. There was no evidence, however, of infection proceeding either from the wound or from the lungs. It must be remembered, however, that it is sometimes impossible to obtain post-mortem bacteriological evidence of a general septicæmia—a fact which shows the importance of a bacteriological examination of the blood during life. It is to be regretted that this was not done in the present case.

Having excluded carbolic poisoning and fat embolism sufficient to cause death, and sepsis, we are left with chloroform as the most reasonable explanation of the changes in the tissues, changes which are evidently the result of an active protoplasmic poison.

We must admit, however, that we are able to instance cases in which the evidence as to the cause of death was strongly in favour of sepsis, and in which the post-mortem findings, as well as the symptoms, were practically the same as those met with in delayed chloroform poisoning.

The best examples of such cases are to be found in deaths after operation for acute peritonitis, for suppurative salpingitis, and for other more or less acute infective conditions within the abdomen.

We shall show microscopical preparations and microphotographs from the heart, liver, and kidneys of a child, aged 6 years, who died nine days after an operation for recurrent perityphlitis. The symptoms in this case were indistinguishable from those which are supposed to be characteristic of delayed chloroform poisoning, and the specimens show changes identical with those which some maintain are characteristic of that condition. The child was operated on during a quiescent period. The omentum was thickened and adherent to the cæcum. In separating it to look for the appendix, two foci of necrotic granulation tissue, mixed with a few drops of inspissated pus, were opened into. The appendix was discovered after considerable search: it was only an inch in length, was only slightly thickened, and contained no concretion. As it did not appear to have been responsible for the perityphlitis, and as its position was such that its removal would have been attended with great difficulty, it was deemed advisable to leave it alone.

Post-mortem examination — External appearances. — Small ulcers and excoriations over the external surface of the upper and lower lips; the teeth are all carious, and the surrounding gums thickly studded with small ulcers exuding pus. Ulcerations in the buccal cavity; no appearance of ulceration or congestion in pharynx.

The abdominal wound, which corresponds in situation and direction to the usual incision for appendicectomy, is healed, with the exception of the middle portion, which is patent, and communicates with a subcutaneous sinus extending downward towards the lower end of the incision. This sinus does not communicate with the abdominal cavity.

Heart.—No increase in the quantity of pericardial fluid. Several capillary hæmorrhages are scattered over the

posterior and upper surfaces of the pericardium and over the base of the heart posteriorly; no pericarditis; no endocarditis. The only noteworthy feature about the heart was that it was rather paler and more friable than normal.

Lungs.—No fluid in subpleural sac, and no pleurisy. Both lungs, but especially the right, show, over their several lobes, numerous subpleural hæmorrhages varying in size and extent. In the lower lobe of the right lung, hæmorrhages in the substance of the organ. The lungs are œdematous, but there is no pneumonic consolidation. The bronchial glands are not caseous. There is no evidence of diphtheritic or other septic inflammation in the larynx or trachea.

Abdomen.—There is no evidence of recent peritonitis, but in the region of the cæcum the bowels are matted together by fibrous adhesions, which are attached to the parietal peritoneum. A piece of the omentum is found to have been stitched over the front of the cæcum, and the wall of the ileum, about three inches above the ileo-cæcal valve, has been transversely inverted for one third of its circumference by a continuous Lembert suture. Neither in this region nor in the neighbourhood of the cæcum or appendix has there been any apparent leakage from the bowels; the signs of acute peritonitis are entirely wanting. The appendix, only one inch long, is somewhat thickened and situated on the posterior aspect of the cæcum. There are no tuberculous glands in the region of the cæcum or elsewhere in the abdomen.

Liver.—The liver is not enlarged; the surface is very yellow and slightly mottled. On section the portal spaces are prominent as dark red lines, but the lobules are of a uniformly pale yellow appearance. The central veins are not visibly distended. There are no areas of focal necrosis; no gall-stones.

The spleen is not enlarged. Its tissue is firm. The Malpighian bodies are prominent.

Kidneys.—The superficial and deep cortex are swollen, and present a uniformly pale yellow appearance.

Alimentary canal.—The pharynx is healthy, but the œsophagus, three quarters of an inch below the level of the larynx, begins to exhibit intense inflammation of its wall, going on to complete necrosis of the mucous membrane. This condition extends throughout the whole of the remaining portion of the œsophagus, but it apparently ceases at the cardiac orifice of the stomach. The necrosed mucous membrane is swollen, varies in colour from dark brown to black, is soft in consistence and easily detachable, and presents an irregular, shaggy appearance. The submucous coat, the muscular coat, and the surrounding fibrous tissue of the mediastinum are swollen and infiltrated with blood.

The *stomach* is filled with dark coffee-grounds, as is also the upper part of the small intestine. The mucous membrane of the stomach is perfectly healthy, as is that of the small intestine, with the exception of the Peyer's patches towards the middle of the ileum, which are slightly swollen. The mucous membrane of the cæcum, of the ascending, transverse, and descending colon, show scattered areas of hæmorrhage, which become more numerous and extensive as we approach the rectum, where the whole mucous membrane is infiltrated with blood. There is, however, no necrosis or ulceration of the mucous membrane of the large intestine.

Brain shows no gross lesion. Bone marrow: there is no increase in its amount, nor in its vascularity.

Microscopical appearances—Œsophagus.—The most interesting point about the œsophagus, besides the necrosis of its mucous membrane and the hæmorrhagic inflammatory condition of its wall, is the presence of certain organisms. Superficially in the mucous membrane micrococci are seen, chiefly streptococci, but these appear to be an accidental contamination. The most striking feature is the presence of long and delicate organisms, which extend more deeply into, and more diffusely throughout the œsophageal wall, and in many situations form definite colonies. This organism is more difficult to stain, and does not retain

the stain in Gramm's method. It corresponds in appearance to one of the mouth organisms, and belongs probably to the leptothrix group. This organism is in all probability the causal factor in the production of the local necrosis of the mucous membrane of the œsophagus and the other more generalised toxic changes. The demonstration of this mouth organism in the œsophagus is of importance in pointing to the probable source of infection, especially as in this case no site of primary infection can be determined in the abdominal cavity.

Examination of rectum.—The mucous membrane of the rectum is widely infiltrated with blood, but not necrosed; no organisms can be demonstrated; the leucocytes, which have emigrated from the blood, are chiefly polymorphonuclear leucocytes.

Liver.—In the fresh frozen section stained with sudan there is a very diffuse fatty degeneration of the liver cells. In many lobules this apparently starts in the central zone around the central vein, where the liver cells in many cases have undergone complete dissolution consequent on a preceding fatty change. In some lobules, however, the intermediate zone shows complete disintegration of the liver cells, exactly analogous to that seen around the central vein in other lobules. Around the portal space only a single layer of liver cells has not suffered in the general fatty degeneration. The fat is in the form of minute granules or large globules, which completely fill the cell. The nuclei of some cells also show the presence of small fat globules. Where the liver cells have been completely disintegrated—that is, in the intermediate zone of some lobules and around the central veins in others—they have been replaced by dilated capillaries. In a paraffin section many of the liver cells around the portal spaces, although they do not show any fatty degeneration, present the appearance of early necrosis. Their protoplasm is hyaline in appearance, and the nuclei of many cells have entirely disappeared.

Kidneys.—In the fresh frozen section stained with

sudan fatty changes are found to be limited to the ascending limbs of the loop of Henle, and to the cells of the straight tubules of the cortex and medulla. No fat can be demonstrated in the cells of the convoluted tubules. In a paraffin section the secreting cells of the convoluted tubules show granular swelling, and many show partial granular disintegration. The lumina of these tubules are dilated and filled with granular detritus.

Heart.—Fresh frozen sections stained with sudan show an extremely diffuse and uniform fatty degeneration of its muscle-fibres. This affects the muscle-cells throughout their entire substance. The fat is in the form of minute granules arranged in definite linear rows. In the paraffin sections the transverse striation of the muscle-cells is seen to be well preserved, and there is no evidence of fragmentation.

Lungs.—The fresh sections fail to demonstrate the presence of fat emboli. Paraffin sections show extensive hæmorrhages into the subpleural tissue and into the surrounding alveoli. There is *no* predominance of polymorpho-nuclear leucocytes in these situations. The minute bronchi exhibit capillary bronchitis. No organisms can be demonstrated in the lung.

How, then, are we to account for such an extreme fatty change in the internal organs? Unfortunately, it is impossible to say how far the organs were healthy before the operation. Possibly a certain amount of degeneration may have been present in the liver as the result of the long-continued absorption from the cæcal region. We are indebted to our colleague, Dr. John Thompson, for specimens showing the same extreme fatty change in the livers of two children (to neither of whom had chloroform been administered), who died, the one aged 10 months, from diarrhœa followed by broncho-pneumonia, the other aged 9 months, after an illness of five weeks, from broncho-pneumonia following influenza. It cannot be denied, therefore, that an extreme degree of fatty change in the liver may occur apart altogether from

chloroform. And if anything like the same degree were present in our perityphlitis case, it is easy to understand how, as Guthrie suggests, chloroform may act as the "last straw." On the other hand, one of us operated recently on a boy suffering from general suppurative peritonitis secondary to a gangrenous appendix, which lay in contact with the under surface of the liver, the cæcum not having descended into the right iliac fossa. In this case the liver, which was exposed at the operation, appeared, as far as could be judged by the naked eye, to be perfectly healthy. The abdomen was drained through four openings and the patient made an uninterrupted recovery, so that unfortunately we had no opportunity of further investigation of the liver. If the heart and kidneys were as healthy as the liver appeared to be, it is quite possible that the chief explanation of the boy's recovery lies in the fact that the toxins were not sufficiently poisonous to set up degenerative changes, and that had the liver been fatty, he would possibly have died.

Turning next to the condition of the heart and kidneys in the child who died after the operation for recurrent perityphlitis, we found that both these organs, especially the heart, showed the same high degree of fatty change as the liver; indeed, one seldom meets with such an extreme and diffuse fatty change in the heart as is seen in this case. It is interesting in this connection to note that in the clinical record of the case special reference is made to the irregularity, as well as to the frequency, of the pulse. It should be mentioned also that there was no albumin in the urine prior to the operation, and that a small amount was present after it. Now, even admitting that there may have been a considerable amount of fatty change in the liver before the operation, it is hardly reasonable to suppose that a child who, prior to the operation, was going about in comparatively good health, could have an equal amount of fatty change in the heart and kidneys. The supposition, therefore, is that such an intense fatty change as these organs presented must have

developed after the operation; and, if so, what we want to know is how far the condition was produced by sapræmia —or by septic infection—and how far by chloroform. We are not prepared to give a definite answer to this question without further investigation.

In the case we are now discussing, although the microscope showed that the heart was intensely fatty throughout, yet to the naked eye the organ did not appear to be much affected, and had it not been for our special interest in the subject of this communication it would probably not have been considered necessary to examine it microscopically. And the same may be said of the kidneys.

The subject appears to us not to have received the attention it deserves, and we think that pathologists would do well to inquire more closely than has hitherto been done into the exact nature and causation of the degenerative changes found in the organs in "surgical" post mortems.

Every surgeon who has closely watched patients after operations in cases of acute appendicitis—and occasionally also after operations performed during the quiescent period—must have observed the critical condition in which he occasionally finds his patient about forty-eight hours or so after the operation. The "chloroform sickness" has persisted, the vomited matter is of the dark hæmorrhagic type already referred to; the patient has become rapidly emaciated; the pulse is small, rapid, and often irregular; there is sleeplessness, often extreme restlessness, and the patient is threatened with death from collapse; the urine contains a trace of albumin; there is no complication connected with the wound, no pain, no increase of tenderness, and no distension; nevertheless, the patient is in a very critical condition, and the surgeon fears either general toxæmia, or the commencement of a spreading peritonitis. Should no improvement set in, the surgeon will probably deem it necessary to open up the wound and investigate the deeper parts. Fortunately, he may find nothing wrong at the seat of operation, and after adopting certain therapeutic measures, he is relieved to

find that during the next twenty-four hours the patient's condition may have improved to such an extent as to call for no further anxiety.

What is the exact cause of the symptoms in such cases? Are we dealing, though in a milder degree, with the same acute fatty degenerative changes that we can demonstrate as having occurred in the fatal cases? and, if so, what share has the chloroform taken in bringing them about? We should be interested to hear if those surgeons who invariably use ether in such cases have had the same experience.

It is not our intention to discuss or compare the relative merits of chloroform and ether, or to throw further discredit on an already too much abused drug. We are of opinion, however, that every endeavour should be made to arrive at a true estimate of the harmful after-effects of chloroform. If it be proved that in effective conditions such as we have been considering chloroform is capable of so adding to the harmful effects already produced by bacterial toxins as to play the *rôle* of the "last straw," the question of substituting ether must be seriously considered even by those who advocate chloroform.

Vomiting.—All authors who have written on the subject of delayed chloroform poisoning draw attention to the severity of the vomiting. Guthrie says vomiting was a marked feature in all but one of his cases; "it was copious, frequent, and persistent, and the vomited matter sooner or later almost exactly resembled the dregs of beef-tea." Our own experience fully confirms these observations. Indeed, we have been so much impressed by the character of the vomiting that we have thought it worth while to inquire somewhat closely into the frequency and circumstance of its occurrence, with the view of throwing more light, if possible, on its true significance, and of ascertaining how far it may be regarded as a measure of the toxic action of chloroform. For the last twelve months the house-surgeons at the Royal Edinburgh Hospital for Sick Children, aided by the ward-sister, have recorded the

nature and frequency of the vomiting on the temperature charts by means of red dots, one for each act of vomiting, small dots being used for retching or slight vomiting, and large dots when it is more copious. More recently we have indicated the beef-tea-like vomiting by black dots. Taking the last 500 operations performed inside the hospital during the past twelve months, Dr. Dunbar, the last house-surgeon, to whom we are indebted for the following statistics, finds that in 10 per cent. the vomiting was of a severe type; that is to say, it was repeated from 8 to 46 times, and lasted from 24 hours to 3 days, and in one or two instances even longer. The vomited matter consisted at first of gastric juice largely diluted with mucus; a little later it contained some yellow or green bile, or was mixed with water or undigested milk, when either of these had been given. At a later stage it generally became more copious, and took the form of a dark green watery fluid, while finally, in the most severe cases, amounting to about 2 per cent. of the total, it became of a dark brown colour and flaky, like thick beef-tea.

As regards the nature of the operation in the 50 cases (10 per cent.) of severe vomiting, it was found that 21 were for tuberculous cervical glands, while the remainder were, for the most part, serious operations, and frequently abdominal. Speaking generally, however, it was found that the severity of the vomiting was to a large extent in proportion to the duration and severity of the operation. A study of the vomiting after the operations for the radical cure of hernia affords conformable evidence of this fact. In children this is a comparatively simple operation, and the average duration of the anæsthesia, including its induction, is only twenty minutes. After the 107 operations for hernia performed during the past year, the vomiting was severe in only 6 cases, and of these 3 were attended with complications necessitating a more prolonged anæsthesia. Another point which these statistics seem to bring out is that severe vomiting is more frequent

in patients suffering from any form of infective disease—whether acute or chronic—than it is in children who are free from such disease. It was found that 90 per cent. of the cases of severe vomiting were operated on for some infective disease, whereas only 42 per cent. of those in which the vomiting was either absent or very slight were for infective conditions. This may partly serve to explain the comparative absence of severe vomiting after operation for hernia. Occasionally, however, striking exceptions occur—to wit, the fact that the two cases which we have here recorded as examples of delayed chloroform poisoning were after operations for uncomplicated hernia and genu valgum.

As to the nature of the dark brown fluid, we may take it that it is due to altered blood, derived in the majority of cases from the capillaries of the gastric mucous membrane. This symptom, like the others, is, however, common to a variety of conditions; for we find it occurring after various poisons taken by the mouth, in acute yellow atrophy, in icterus gravis, in various septicæmias, amongst which may probably be included the majority of the cases of so-called post-operative hæmatemesis. We have already shown that it may be produced experimentally by the subcutaneous inoculations of chloroform, even in small doses, and Ungar has produced severe gastro-adenitis in dogs by repeated inhalations of chloroform.

But here, again, we meet with the initial difficulty in distinguishing the hæmorrhagic form of vomiting occurring in delayed chloroform poisoning from that which not infrequently occurs in septicæmia. If the latter complication can be definitely excluded, we are left with chloroform as the only reasonable explanation.

How, then, is the hæmorrhage actually produced? At first it may be supposed that it is the direct mechanical result of the repeated and severe retching, an explanation which, we venture to think, has been too readily accepted. Two strong arguments can be brought against this view,

viz., (1) That hæmorrhagic vomiting is by no means preceded by frequent or violent retching; (2) that very violent retching is often seen in severe bilious attacks without a trace of blood in the vomited matter. By some it has been suggested that the hæmorrhage is the result of an acute gastritis due to the swallowing of a certain amount of chloroform vapour. But, as has also been seen from the experiments on rabbits, a local lesion in the gastric mucosa may be due to chloroform absorbed by the blood and excreted into the stomach by the gastric glands. This appears to us to be the most reasonable explanation of the petechial gastric hæmorrhages. We must remember, however, that severe parenchymatous changes in the liver must almost of necessity, by interfering with the circulation, lead to increased hyperæmia of the gastric mucosa, which in its turn favours the occurrence of capillary ecchymosis. This hæmorrhagic form of vomiting is almost invariably associated with grave constitutional disturbances, as evidenced by the collapse, rapid and feeble and sometimes irregular pulse, restlessness, and frequently also cerebral excitement, a combination of symptoms which point to a general toxic condition due either to organismal infection or to an auto-intoxication set up by the chloroform.

Experimental Observations.

Fourteen experiments have been performed to demonstrate the action of chloroform on the tissues, when induced by subcutaneous injection, and when administered in the ordinary way by inhalation.

The experiments have been performed on rabbits. Of those fourteen experiments, only six will be referred to at present. The method of subcutaneous injection was adopted in order to demonstrate the full action of the drug on the tissues.

Experiment 1.—A rabbit weighing 5 lbs. 2 oz. was *injected subcutaneously* with 2 c.c. of pure chloroform. In a few minutes the animal became extremely drowsy, and

for some hours after was manifestly under the action of the drug. In the evening it had perfectly recovered, was moving about freely, and took food. Next morning the animal looked perfectly well, and ate freely. Twenty-four hours after the injection it weighed 4 lbs. 14 oz. On the evening of the second day it became dull and apathetic, and on the third morning it was manifestly ill. It refused food, and lay in the corner of the cage. At 10 o'clock the same evening the condition was unchanged, and on the following afternoon it was found dead, about seventy-five hours after the injection. Post mortem same day. Marked diminution in amount of subcutaneous and omental fat. *Stomach* distended with partially digested food. Numerous petechial hæmorrhages in the gastric mucosa.

Heart.—Right side of the heart flabby, distended with dark fluid blood. On section nothing abnormal beyond pallor of the myocardium. Right lung showed patches of broncho-pneumonic consolidation. Left lung nothing abnormal.

Liver markedly fatty, the fatty change being apparently most marked in the central and intermediate zones, the portal tracts and peripheral zones appearing red and congested.

Spleen.—Nothing abnormal.

Kidneys.—Nothing abnormal, beyond pallor.

Histology—*Heart.*—Well-marked fatty degeneration of the myocardium. In places the transverse striation almost completely lost. In places there was also some tendency to transverse segmentation of the fibres. Some of the fibres rather granular and swollen. The nuclei throughout stained well.

Lungs.—Sections from the right lung show an acute broncho-pneumonia. No fat could be demonstrated in the lung capillaries or alveoli.

Liver.—An intense degree of fatty change, especially in the central and intermediate zones. Many of the liver cells towards the centre of the lobule completely disorganised. The cells were full of minute droplets not

tending to coalesce. Further out in the lobule the globules are large. The narrow zone of liver cells at the periphery of the lobule showed no fatty change at all. The nuclei of many of the cells in the central zone stained very badly. No fat was demonstrated in any of the hepatic vessels (Plate II, fig. 3).

The kidneys.—Both show well-marked cloudy swelling of the convoluted tubules and ascending loops of Henle. Well-marked fatty change in the epithelium of the collecting tubules, and small irregularly scattered foci of fatty change in the epithelium of the ascending loops of Henle, and in a few convoluted tubules. The *glomeruli* throughout show no change, and no fat was demonstrated in any of the vessels (Plate II, fig. 4).

Experiment 2.—As a contrast experiment, a rabbit weighing 4 lbs. 12 oz. was injected subcutaneously with 3 c.c. pure ether. The animal suffered no inconvenience beyond the temporary intoxication produced. Next day it had recovered perfectly. It was killed 75 hours after the injection, and naked-eye and microscopical examination of the various organs showed nothing abnormal.

Experiment 3.—Rabbit, weighing 3 lbs. 12 oz., injected with 2 c.c. pure chloroform. Recovered completely from the intoxication and on the following morning looked perfectly well. Its weight 24 hours after the injection was 3 lbs. 10 oz. On the following day 3 lbs. 3 oz. and very dull and apathetic. Died during the following night, between 80 and 90 hours after the injection.

Post mortem.—Marked diminution of the subcutaneous, omental and peri-renal fat. A few petechial hæmorrhages in the gastric mucosa. Heart-muscle extremely pale. Nothing else abnormal. Lungs showed nothing abnormal.

Liver intensely fatty, the appearances of the liver in experiment 1 being exactly reproduced.

Kidney.—Marked pallor: no evidence to the naked eye of fatty change.

Microscopic Examination. *Heart.*—Fatty change not marked; a few globules in some of the fibres. The

DELAYED CHLOROFORM POISONING.

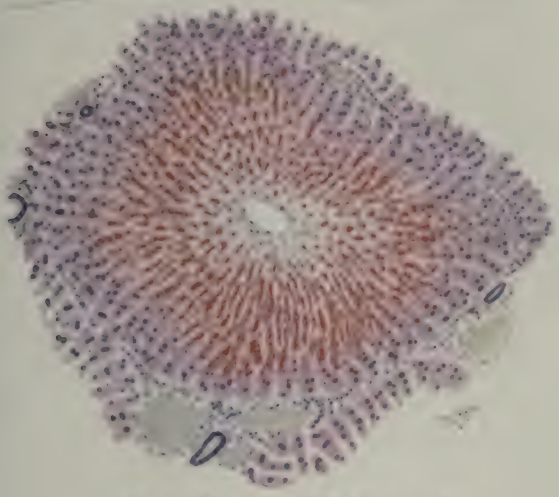

Fig. 3. Hæmatin and Sudan 3 × 80.

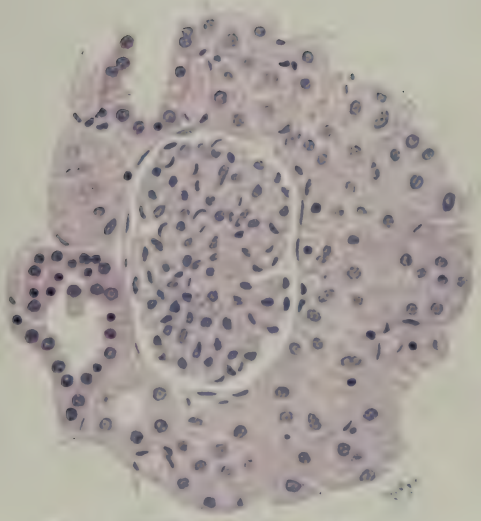

Fig. 4. Hæmatin and Eosin. × 250.

To Illustrate Mr. Stiles and Dr. Stuart M'Donald's Article.

Liver and Kidney of Rabbit (Experiment I.)

muscle-fibres swollen, granular, and showing some loss of transverse striation. Nuclei stained rather irregularly at places; no transverse segmentation of fibres.

Liver.—Well-marked degeneration of cells in central zone. Practically all the cells containing small globules of oil, and the nuclei of many of those cells staining very poorly. As in experiment 1, the extreme outer part of the peripheral zones showed practically no fatty change, but the cells were granular and swollen.

Kidneys.—Only a comparatively slight degree of fatty change in the collecting tubules, with scattered foci in the cortex as in experiment 1. The convoluted tubules, however, show well-marked cloudy swelling, amounting in places to an almost complete necrosis.

Experiment 4.—Rabbit, weighing 4 lbs. 12 oz., injected with 4 c.c. of pure ether.

The animal again suffered no inconvenience beyond the temporary intoxication. It was killed 96 hours after the injection and naked-eye and microscopic examination revealed nothing abnormal.

Two experiments have been performed on rabbits with the special view of determining the action of the chloroform on the liver, the drug being administered by inhalation in the ordinary method. As control-observations and in order to determine the condition of the liver at the time of operation the experiments were begun by excising a small piece of liver under ether anæsthesia.

Experiment 5.—A rabbit, weighing 5 lbs. 4 oz., was anæsthetised with ether and a small piece of liver removed. There was practically no hæmorrhage. The abdominal wound was stitched up and covered with collodion. The operation lasted twenty-five minutes. Seven days later the wound was examined and found to be perfectly healthy and with good union. The animal was then deeply anæsthetised with chloroform in the usual way and was kept continuously under for about twenty-five minutes. The animal did not take the chloroform well and partial recovery of consciousness was allowed several

times. Three hours afterwards the animal had recovered perfectly and next day was quite well. Forty-eight hours after the chloroform narcosis the animal was killed and a post mortem at once performed. The liver was found to be slightly paler than normal, and there was some slight mottling, there being a slight yellowish tint towards the centres of the lobules. The other organs showed nothing abnormal to the naked eye.

Histology—Liver removed at the first operation.—In the majority of the sections the liver cells showed nothing abnormal. Here and there some small oil globules were present in the cells. This was not seen in any particular zone, and nowhere was it marked. Many fields showed absolutely no trace of this fatty change.

Liver removed at post-mortem.—Paraffin and frozen sections were prepared and the latter were specially stained for fat with osmic acid and sudan. In contrast to the appearances before the chloroform, there was now seen to be an extensive fatty change in practically every lobule in the liver. The change was most marked in the intermediate and central zones, a few rows of cells indeed next the portal tract showing no fat whatever. These latter cells, however, were distinctly granular. The nuclei of the liver cells throughout stained somewhat feebly, not more so, however, in the intermediate and central zones. In those latter situations the liver cells were crowded with small droplets of oil which did not tend to coalesce. The protoplasm of those cells, too, was also very distinctly granular. The condition did not approximate in intensity to the degree of the fatty change seen after the subcutaneous injection of the drug, but the difference appeared to be purely one of degree.

Heart.—Beyond some slight granularity of the muscle-fibres, in places, sections showed practically nothing abnormal. In none of the sections could any fat be demonstrated.

Kidney.—Sections showed distinct cloudy swelling of the convoluted tubules, and an occasional convoluted tubule

showed very distinct fatty change. In the collecting tubules there was also some distinct fatty change; the nuclei everywhere stained well, and the glomeruli showed nothing abnormal.

Experiment 6.—This experiment was started in the same way. A rabbit, weighing 2 lbs. 5 oz., was anæsthetised under ether, and a small piece of liver removed. The wound was stitched up and chloroform was substituted for the ether, the animal being kept continuously and fairly deeply under for forty-five minutes. The animal recovered very slowly from the narcosis, being drowsy and semi-conscious several hours. On the next day, however, it had recovered perfectly. It was killed forty-eight hours after the original operation.

Post-mortem.—Beyond slight pallor of the liver and kidneys practically nothing abnormal was noticed.

Liver excised at beginning of operation.—Some distinct fatty change was demonstrated in the intermediate and central zones and in a few places here and there in the peripheral zones. The change was nowhere well marked. Otherwise the liver showed nothing abnormal.

Liver obtained at post mortem.—Here a condition similar to that before operation was noted, but the fatty change was very distinctly more marked. This case, however, has not been studied in detail, as the fatty change present before operation seems to vitiate the experiment. It may be stated, however, that no fatty change could be demonstrated in the heart-fibres, though in the kidney the fatty change in the collecting tubules was fairly well marked. The convoluted tubules showed cloudy swelling, but no fatty change. An explanation of the fatty condition found in the liver in this case may be found in the fact that the animal was rather small and poorly nourished. In our future experiments we shall endeavour to eliminate this fallacy by using only well-grown and well-nourished animals; and we shall also endeavour to exclude any fallacy from a possible collection of fat in the liver cells during digestion.

That fatal cases of delayed chloroform poisoning are exceedingly rare may be gathered from the fact that so few cases have been recorded. Even granted that the condition may have been overlooked or misinterpreted, we cannot overlook the fact that surgeons of large experience who recognise the condition state that they have only met with, perhaps, a single case (Mikulicz). Even in children, in whom the condition is probably more common, our own experience is limited to only three, or at most four, cases out of 3000 operations performed by one of us during the past five years in the In-patient Department of the Royal Edinburgh Hospital for Sick Children. The rarity of the condition seems to indicate that there must be some predisposing factor in addition to the chloroform, which, in the light of our present knowledge, must be regarded as the existing cause. This predisposing factor must be looked for in some already existing morbid condition in the body or in some special idiosyncrasy to the drug. The morbid changes of the liver are so striking, and the fatal issue takes place so soon after the administration of the chloroform, that the idea of a pre-existing disease of the liver has suggested itself to some. So long ago as 1860 Symonds of Oxford drew attention to the danger of chloroform in patients suffering from fatty liver. Guthrie holds a similar view, but insists that "in children the form of fatty liver to which attention has been drawn is altogether peculiar to these cases of fatality following operation under chloroform." He states that he is familiar with most kinds of fatty liver, but that he has never seen the pale fawn or buff-coloured variety except in such cases as these. His distinction is that in these cases the fatty change affects the entire organ and is not confined to portions of it. With all due deference to Dr. Guthrie, one of us, as a result of a considerable post-mortem experience, is of opinion that the same colour and the same diffuse fatty change may be met with in the liver in conditions apart altogether from chloroform. We are quite prepared to admit that a pro-

fuse morbid condition of the liver may play some part. Indeed, we may briefly refer to a case in point, viz., that of a boy, aged 7 years, who died from collapse on the fourth day after the administration of chloroform for the purpose of examining a stiff knee-joint, secondary to specific disease of the lower end of the femur. Before the anæsthetic was administered it was observed that the liver was considerably enlarged. The anæsthesia, including its induction, only lasted a quarter of an hour. No incision was made, nor was there any attempt made to forcibly straighten the knee. The patient came well out of the anæsthesia, but was very sick the whole day. On the day following the vomited matter was dark brown and distinctly "*coffee ground*," and this persisted in spite of everything until death. At first the patient was very restless, but subsequently he became more and more collapsed, and died in a semi-comatose condition.

Post-mortem examination. (1) *Liver.*—The appearances closely resemble those seen in the patient on whom osteotomy was performed. There is, however, a well-marked fibrous overgrowth of Glisson's capsule, and there are numerous collections of lymphocyte-like cells here and there in the portal tracts. There is, however, no distinct intercellular cirrhosis. The vessels are thickened throughout. Sections stained with sudan show an extreme degree of fatty change throughout the entire hepatic lobule. The peripheral zone shows well-marked fatty infiltration, many of the cells being entirely filled with large globules of fat. The nuclei are displaced, but stain perfectly well. In the intermediate and central zones the fat is in much smaller droplets and the cells are not increased in size—some indeed are markedly diminished. The nuclei are again well stained. There is some dilatation of the capillaries round the central venule.

2. *Kidney.*—Unfortunately, only a paraffin section was available for examination, and the fat of course cannot be satisfactorily demonstrated. The cells of the convoluted tubules and the looped tubules of Henle showed

well-marked cloudy swelling. The vacuolated appearance of the cells strongly suggested a fatty change. For purposes of comparison sections of the kidney in the osteotomy case were prepared in an exactly similar manner, and the appearances were almost identical with those seen in the present case.

We do not think that Dr. Guthrie has brought forward sufficient evidence that the fatty change he describes is the essential predisposing factor, the chloroform only acting as the "last straw." On the contrary, we are by no means convinced that the fatty change may not be entirely due to the chloroform acting on a previously healthy liver. Indeed, experimental evidence seems to give strong support to this possibility. Moreover, while the changes in the liver are so striking, it must not be forgotten that other organs give evidence of a general toxic poisoning, and it seems hardly justifiable therefore to attribute the fatal result to hepatic insufficiency.

In addition to a pre-existing morbid condition of the liver, other authors have sought for the explanation in organic disease of the other viscera. Thus, disease of the kidneys, vascular system, etc., have all been regarded as possible pre-existing factors. While it is impossible to deny that these may play some part, it is well known that subjects of grave organic disease often stand chloroform so well that we cannot regard the pre-existing lesions as essential causal factors. Again, it must be remembered that in some of the fatal cases organic disease can be definitely excluded, so that we are driven to the other possibility, viz., that the essential predisposing factor is to be sought for rather in some special idiosyncrasy to the action of the drug.

It will, we think, be admitted that various degrees of fatty change in the liver may occur without giving rise to associated signs and symptoms which can be recognised clinically. In patients the subject of chronic disease, *e.g.* tuberculosis, a certain amount of fatty change is frequently present in the liver, but we are unwilling to admit that

anything like the degree of fatty change found in the liver at the post mortem in cases of delayed chloroform poisoning is at all likely to have been present in patients who, apart from such affections as uncomplicated hernia, genu valgum, etc., appear to be perfectly healthy.

It is more than probable also that idiosyncrasy plays an important part in many of the fatalities during the administration of the chloroform. Admitting this idiosyncrasy, however, it is worth while considering the manner in which chloroform brings about a fatal issue in the delayed deaths.

As we have seen, some would attribute this to a direct destructive action on the red blood-corpuscles leading to deficient oxidation, which in its turn might explain the increased deposit of fat in the organs. There does not seem to be sufficient evidence in support of this view. The most reasonable explanation of the condition is to be sought for in some intoxication, though the exact manner in which this is brought about is still obscure. The experimental results on the effect of chloroform clearly show that it is an intense protoplasmic poison capable of producing changes in the organs sufficient to interfere seriously with their function. We have seen that the most prominent histological feature is a fatty change involving especially the liver, kidneys and heart, and probably also elsewhere. It is difficult to say, however, whether the fatty change in the liver is to be regarded as of the nature of a degeneration or an infiltration. Undoubtedly the appearances in many cases are those of the local formation of fat from the cell protoplasm, but recent researches have shown that many of the conditions formerly described as degenerations are really of the nature of infiltrations, the fat being derived from other parts of the body and elaborated from the blood by the liver cells.

The symptoms in cases of delayed chloroform poisoning closely resemble those met with in other auto-intoxications—such, for example, as diabetic coma, a condition now

generally attributed to the production of certain acids, acido-acetic acid, etc. It is worthy of note that many observers have detected a great increase of acetone in the urine after chloroform anæsthesia, and it seems reasonable to suppose that very possibly the blood may contain some of the poisonous precursors of acetone, and that in certain cases these are produced in so great an amount as to determine a condition analogous to diabetic coma. That there is a possible relationship is further suggested by the very striking diminution of fatty tissue throughout the body in rabbits poisoned by the subcutaneous injection of chloroform. It must be remembered, however, that the same rapid emaciation is also observed in animals poisoned by bacterial toxins.

The most recent physiological researches on the source of acetone in the body have disproved the old idea that it is derived from either proteids or carbohydrates, and should show that fat is its true source. We should like it to be understood, however, that we regard the analogy between delayed chloroform poisoning and diabetic coma merely as a working hypothesis, and we think the question is worthy of further investigation. If an excessive acetonuria be shown to exist in delayed chloroform poisoning, we might hope to find in it the means of recognising milder degrees of the intoxication, and possibly also of differentiating the condition from such post-operative complications as delayed shock, sepsis, etc.

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34. Waldvogel, 'Langenbeck's Archiv,' vol. lxvi, 1902.

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(*Read May the 6th*, 1904.)

Discussion.

The Chairman (Mr. Watson Cheyne), in the name of the Society, thanked Mr. Stiles very much for the most admirable address which he had given them, and for calling attention to these peculiar cases. He had to confess, however, that he was still not quite convinced that the chloroform was the only causative factor in these cases, and that they must be looked on as after-effects of the administration of that anæsthetic. Although he had had two or three cases at Paddington Green Children's Hospital, Mr. Cheyne thought it very remarkable that he had never met with similar occurrences either at King's College Hospital or in private practice. The extremely short administration in some of the cases, for example, tenotomy, and the minute dose administered, made it very difficult to believe that the chloroform was the cause of the fatal result. He would await the publication of the paper and the possibility of studying the facts more carefully in detail with the greatest interest.

Dr. Leonard Guthrie thought that the frequency of these fatalities following operations under chloroform upon children might be greater than was indicated by the number of cases recorded in this country. Ten years previously he had published in 'The Lancet' a series of ten cases, nine of which had happened at the Children's Hospital, Paddington Green. Since then, three more had occurred at the same institution. He doubted whether such experience was unique. Possibly a search in the archives of other children's hospitals might reveal more cases of a similar nature. He did not believe that they were attributable to septic infection of operation wounds. Yet the symptoms, which resembled those of acute yellow atrophy of the liver, pointed to toxæmia of some kind. The view that fat embolism was the cause of death did not accord with the symptoms, pathological findings, and nature of the operations. The case against

chloroform was that prolonged and repeated inhalation of that anæsthetic produced in animals profound fatty changes, not only in the liver but in the kidneys and other organs, and that such changes were almost constantly found in children dying in the manner described after operations under chloroform. But on the other hand it was difficult to conceive that such extreme fatty changes could be induced by the small amounts of chloroform absorbed during a period of anæsthesia which in some cases did not exceed a quarter of an hour or twenty minutes. Hence he inclined to the view that a fatty condition of the liver was pre-existent at the time of operation in such cases, that chloroform by its deoxidising powers aggravated and intensified the fatty changes present, and thus rendered the liver incapable of dealing with toxins entering the portal system from the intestines. The result was that such toxins passed into the general circulation and could not be eliminated on account of the specific action of the chloroform on the kidneys. The *rôle* of the chloroform was therefore that of the "last straw." The existence of such fatty livers might be suspected though it could not be proved by the history of so-called "bilious attacks," characterised by vomiting, headache, epigastric pain, diarrhœa, or constipation, associated with the odour of acetone in the breath and urine. When such a history was obtained, ether should be the anæsthetic used, as experiments on animals showed that ether possessed the power of inducing fatty changes to a far less extent than chloroform.

Dr. F. W. Hewitt (introduced) had never seen any cases at all comparable to these. He supported Dr. Guthrie's request for an inquiry, on account of the extreme importance of the subject to the profession. He wished to ask Mr. Stiles what method of giving the chloroform had been employed, because, although most improbable in these instances, he thought that sometimes acute infections obtained access to the body by chloroform inhalation. He suggested that one of the children's hospitals change its customary anæsthetic for one or two years to a mixture of chloroform and ether, in order to observe the results.

Mr. F. F. Burghard remarked that in one of Dr. Guthrie's cases at Paddington Green, on which he had operated, the notable thing was that death occurred after the *second* time of operating. The case was one of a very large nævus of the chest and arm, and the first operation, which had taken place several weeks before, was much the longer and more severe of the two; nevertheless the child recovered without a symptom of any sort referable to chloroform administration.

Dr. J. F. Silk (introduced) confessed that he had been very sceptical with regard to Dr. Guthrie's first paper ten years ago; he was now very astonished to find that it had been discovered in Edinburgh, of all places, that quite possibly chloroform might produce some ill effects. He commented on the occurrence of

the condition only in older children, in spite of the fact that chloroform was given to very large numbers of infants. It was strange also that no relation could be established between the size of the dose administered and the production of the condition. He considered that, in Mr. Stiles' experiments, 2 c.c. subcutaneously was too big a dose for an animal weighing three pounds, and was not surprised to hear of their succumbing; that was not a fair test.

Mr. H. P. Noble (introduced) had administered two of the anæsthetics in Dr. Guthrie's cases. Perhaps these cases were commoner than was usually supposed. He now thought that two deaths he witnessed at Great Ormond Street, when house-surgeon there, were probably due to this condition.

Mr. W. J. McCardie (Birmingham) (introduced) had never seen a case, although he remembered the death of a patient on the fourth day after similar symptoms; however, no autopsy took place. Froster had described two cases of parenchymatous degeneration due to chloroform administration, and showing slight albuminuria, vomiting, and collapse before death. The first case died on the second day, and the second, a girl of eleven, on the fourth. Fatty changes were found in the heart, liver, and kidneys. That author lays stress on the kidney lesions, especially on the fact that they were more advanced in the tubules than in the glomeruli, which was a diagnostic feature. He wondered whether such conditions were more apt to occur in lymphatic children, as they stood most adverse influences worse than other children. The moral was to always use ether in children.

Mr. H. J. Stiles (Edinburgh), in reply, said time did not permit of his going into the literature of the subject. What had struck him most about the German cases was that the majority were reported from gynæcological clinics, and occurred after operations for septic conditions within the abdomen. The possibility of a severe septic intoxication in these cases was so great that it was doubtful how far it was justifiable to attribute the deaths to delayed chloroform poisoning. He deprecated the practice of examining gynæcological cases under chloroform for diagnostic and demonstrative purposes, and then giving the drug again a day or two later for the operation. He advised that careful autopsies and microscopical examinations should be made in all cases in which there was any doubt as to whether the patient had died from sepsis or from delayed chloroform poisoning. It was important also to compare such cases with those in which death had occurred from septic disease without operation. He inclined to the belief that two factors were often at work, viz. a bacterial toxin and the chloroform. In answer to Dr. Silk, he suggested that delayed chloroform poisoning might easily be overlooked in infants who had undergone minor operations such

as circumcision, etc., because such cases were invariably treated as out-patients. With respect to the experimental dose, he did not consider that the injection of 2 c.c. was excessive, although this amount was purposely employed to bring out the full action of the drug on the tissues.

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73. A CASE OF PERFORATED GASTRIC ULCER IN A BOY AGED 13 YEARS; DISEASED APPENDIX; OPERATION; RECOVERY.

By W. WATSON CHEYNE, C.B., F.R.S.,

AND

HAYDOCK WILBE, M.D.

(Introduced.)

On March the 16th, 1904, the patient, a boy aged 13 years, who had previously been perfectly well, complained of some stomach-ache during the afternoon when returning from school. In the evening this became worse, and he asked to be allowed to go early to bed; his mother put a hot-water bottle over his stomach, which relieved him in a few minutes. He got up on the morning of March the 17th much better and practically felt well except for a little abdominal pain when stooping; the exact position of the pain was not well defined. He, however, asked to be allowed not to go to school. In the morning he had some Eno's fruit salt and ate a very good dinner that day. About an hour after dinner he began to complain of severe abdominal pain, and very soon became seriously ill. Dr. Wilbe was sent for and saw him between three and four o'clock in the afternoon, and found him complaining of violent pain in the abdomen which he could not localise very precisely, but he placed his hand towards the right side and about the umbilicus.

He found him very collapsed, with a pulse of about 130, with marked rigidity and tenderness over the whole abdomen. The symptoms were especially marked towards the right and upper part of the abdomen. There was no vomiting. The temperature was normal. Mr. Watson Cheyne saw the patient at 8.30 in the evening. At that time the boy looked extremely ill; there was no abdominal respiration; the alæ nasi were moving; there was marked rigidity of the whole abdomen and general tenderness, but no abdominal distension. The pain and rigidity were not well localised, but were, on the whole, most marked towards the upper part on the right side. The abdomen was tympanitic everywhere, but there was no distinct obliteration of the liver dulness. There had been no vomiting. The temperature was normal. The conclusion come to was that there had been a perforation of some abdominal organ, and from the general history and symptoms pointing more towards the right side, and the probability at that age that the trouble was of the form of a fulminating appendicitis, an incision was in the first instance made over the seat of the appendix. On exposing the peritoneum it at once bulged out in the wound, and on puncturing it gas whistled out. On enlarging the opening the small intestines were forced out, partly by the gas and partly as the result of the great rigidity of the abdominal walls, which had not become relaxed under the anæsthetic. Some thin whitish fluid also came out of the abdomen. For the most part the intestines were normal in appearance, but on some of the coils of the small intestine, which came apparently from the left side of the abdomen, there were some flakes of lymph. The pelvis and lower part of the abdomen were well irrigated with salt solution till the fluid returned clear, and the intestines were then, gradually but with considerable difficulty, returned to the abdominal cavity. The cæcum and appendix were now pulled out. The appendix was long, thickened, a good deal reddened, and contained a large concretion; but there was no perforation

of the appendix nor of the cæcum in the neighbourhood. The appendix was removed in the usual manner.

As it was evident that there must be some graver lesion than the condition of the appendix to account for the state of matters, and as practically the whole of the small intestine had been seen, and as also there was no fæcal material present in the abdominal cavity, a second incision was now made over the stomach. At first nothing seemed to be amiss. The body of the stomach and the pyloric end seemed perfectly healthy; the region of the duodenum was examined; the stomach was turned over and the posterior surface palpated, and still no perforation was found. On further examination of the anterior surface high up towards the cardiac end some turbid fluid and gas bubbled up. After wiping that away and directing a good light into the cavity high up under the diaphragm, a round perforation was found on the anterior surface of the stomach about an inch from the cardiac end which would have admitted a small slate pencil. From this perforation gastric juice and gas were escaping. There was no thickening around; it was a clean punched-out hole, and all the structures in the neighbourhood seemed perfectly healthy. The edges of the ulcer were inverted with a row of catgut sutures, and a second row was then put outside. The peritoneal cavity in the neighbourhood was thoroughly sponged out and the whole cavity again washed out with salt solution. A drainage-tube was passed down towards the seat of the perforation, another towards the left loin, and another (from the lower wound) towards the pelvis. The rest of the wounds were stitched up. The boy stood the operation very well, and his pulse after it was 120. On the night of March the 18th the wounds were dressed. There had been very little discharge. The abdomen was very much distended. The pulse was 130, and the temperature was 99·2° F. There was a good deal of tenderness everywhere. No flatus had been passed. There was very little sickness. The boy had not slept and was somewhat excited. On

the whole he looked rather better than he did on the previous day. He had been having rectal feeding with sips of water by the mouth. On the 19th the abdomen was still in much the same condition, but the patient did not seem any worse than he was on the 18th. The pulse and temperature were the same. There was less tenderness at the lower part of the abdomen, and there had been no vomiting. A little flatus had been passed but not enough to give him relief. Enemata had been given but without any result. At our visit in the evening we gave him an enema again and evacuated some fæces and flatus, which relieved him very much. The drainage-tubes were pulled out and the greater portion cut off, leaving only a small part projecting into the peritoneal cavity. There was very little discharge. On the 20th the patient was distinctly better. The abdomen was not so tense but still very much distended. He had passed a little flatus and slept a little during the night. The tubes were left out. There was very little discharge. He began to take small doses of salines and salol. On the 22nd the patient had a good action of the bowels in the afternoon, and the abdomen was much less distended. He was very much better. On the 23rd feeding by the mouth was begun. From this date the boy went on uninterruptedly well. Solid food was allowed on April the 1st. The wounds had completely healed on the 13th. The patient was allowed up for the first time on the 14th, and went out in a bath-chair. On the 25th the boy was in high spirits, and had apparently perfectly recovered.

The interest in this case lies, in the first place, in the occurrence of a perforating gastric ulcer in a boy aged 13 years. We can find no record of perforation of the stomach in a male at such an early age. In this case the ulcer was typically a peptic ulcer. Gastric ulcers have been found in younger children, but apparently only of a tuberculous character. Thus Dr. G. F. Still * recorded five examples of tuberculous ulcers of the stomach in children, in three

* 'Brit. Med. Journ.,' May the 6th, 1899.

of which histological and bacteriological examination had been made with positive results. In all cases the ulcers were small. The first was a child, aged three and a half years, who was admitted with vomiting and ascites due to tuberculous peritonitis; the second was a child, aged ten months, suffering from cough and tuberculous meningitis, and, as shown after death, from generalised tuberculosis; the third was a case of tuberculous peritonitis; the fourth was a boy, aged nine years, who was also affected with tuberculous peritonitis; and the fifth was a child, aged two years, suffering from the same disease. The author had only found 5 cases out of 226 necropsies. There were no clinical symptoms associated with them. In one of the five cases perforation had occurred but extravasation had been prevented by adhesions. In the above case there was no sign whatever of tuberculosis either about the stomach or the abdomen generally, nor was there any reason for suspecting it. As to the cause of the ulcer in this boy we cannot make any suggestion, and there were no previous symptoms whatever leading to a suspicion that anything was wrong with the stomach.

The second point of interest is that, partly as the result of the symptoms pointing somewhat towards the appendix, and partly on account of the great rarity of the actual condition, the appendix was in the first place cut down upon and a *diseased appendix* was found. The condition of the boy was so extremely bad that one was very much tempted to hope that that might be the cause of the whole trouble; but the presence of free gas in the abdomen made it absolutely certain that perforation of a hollow viscus other than the appendix must have taken place, and the absence of fæcal material in the abdominal cavity, and the fact that practically the whole of the small intestines had passed under review when they were forced out of the wound, led us to assume that this perforation must have taken place either in the stomach or duodenum.

The third point of interest is the situation of the ulcer. It was situated so high up, and was so difficult of access,

that its presence was nearly overlooked. Before it was found the whole of the lower part of the stomach had been examined, the duodenum had been investigated, the stomach had been turned up and the posterior part searched; and had it not been that we were convinced that there must be a perforation, and that a further search was made towards the upper part of the stomach, the condition might have been overlooked.

(*Read May the 27th*, 1904.)

Discussion.

Dr. Porter Parkinson desired to add another case of gastric ulcer to those mentioned by Mr. Cheyne, namely, one which he himself showed at the Society, and which was recorded in the Reports, vol. i.* It was in a child two years and two months old, who had gastric symptoms of an indefinite character. After being in the hospital three or four days it suddenly became collapsed, and there were signs of peritonitis. The gastric ulcer was seen for the first time post mortem, and was on the middle part of the small curvature of the stomach, and perforated at its base. There was also general peritonitis. The post-mortem examination was thoroughly made, and there were no tubercles to be found in any part of the body, nor any evidence of tuberculosis. One of the speakers at the meeting in question suggested that the ulcer might have originated in an inflammation around one of the follicles of the stomach. It was, as far as he could ascertain, one of the youngest cases of gastric ulcer on record.

Mr. Clement Lucas regarded the communication as an exceedingly interesting and valuable one as an instance of gastric ulcer in a small boy. The author had alluded to the paper, with which most of the members were familiar, by Dr. Still, recording similar cases, in which the ulcers were undoubtedly tuberculous, and although Mr. Cheyne said there was no evidence of any tuberculous disease about the abdomen, it still seemed possible that the disease might be tuberculous. He would conceive it possible that tuberculous disease might attack the stomach wall and cause perforation without giving rise to any general tuberculous disease of the abdomen by which it could be well diagnosed. He would like to hear the author's suggestion as to its cause if it were not tuberculous. With regard to the material used by Mr. Cheyne for his sutures, he seemed to have used catgut for both the deep and the Lembert sutures. He, Mr. Lucas, had for some time given up the use of catgut, because, for one reason, he

* *Vide* 'Reports,' No. 50, p. 143, vol. i.

had found it so clumsy, and he did not think it was always reliable. Consequently he was in the habit of using well-boiled silk. For the rest, he congratulated Mr. Cheyne on the result of the operation, especially bearing in mind that the incision over the appendix showed that it was not the appendix which was at fault, and a second operation had to be performed. It would be a valuable paper for the 'Reports.'

Mr. Watson Cheyne, in reply, said he could find no evidence of tuberculosis elsewhere. He did not read the account of Dr. Still's cases, but he had it with him, and found that in every one the tuberculous ulcer of the stomach was associated with tuberculous disease elsewhere. The first had tuberculous peritonitis, the second had tuberculous meningitis, the third had tuberculous peritonitis, the fourth tuberculous peritonitis, and the fifth tuberculous peritonitis. Of course that did not say that his case might not be one of primary tuberculous ulcer of the stomach; but it was a clear punched-out ulcer exactly similar to those met with in adults, and with no adhesions round about. The boy was well looked after in the matter of his food, and why an ulcer should develop in that way was not clear. The point of interest to him was the great diagnostic value of gas in the abdomen. The question of sutures was rather a long one to enter into, but he was still Tory enough to believe that catgut was the best for buried sutures if only they were properly prepared by Lister's formula. Catgut held for a long time but did not hold for ever. And if there was likely to be sepsis in the case the catgut was likely to rapidly disappear. He never used silk for the union of intestines, although he often used it in other tissues where he desired the suture to remain for a long time. He believed the cause of a good deal of the trouble arising so frequently from catgut was because it was not prepared according to Lister's formula.

74. MULTIPLE GLIOMATA OF THE SCALP SECONDARY TO BILATERAL GLIOMA OF THE RETINA.

By EDMUND CAUTLEY, M.D.

This girl is just two years old. She was admitted into the Belgrave Hospital for Children under my care on May the 6th, 1904, for multiple swellings on the head, stated to have only been noticed for a week. The family history is unimportant, but her past history is of the

utmost value. At the age of one year she had a large abscess in the neck on the right side. This opened and healed. Then the right eye "turned in," and eventually became inflamed and bloodshot. She was taken to the Royal London Ophthalmic Hospital and on September the 29th, 1903, the eye was removed. The following pathological report was kindly sent to me from that hospital: "Glioma exophytum. Œdema of the cornea. Anterior chamber contains blood and the angles full of glioma. White nodules on iris near pupil. Iris covered with glioma. Lens *in situ*, but degenerated. Retina detached. Large mass of glioma near optic disc; many satellites behind retina and on choroid. Choroid degenerated."

The child is backward, cannot stand or walk, has only fourteen teeth, and presents slight rachitic changes. The face is pale and looks unhealthy. The socket of the right eye appears healthy. The upper eyelid shows some purplish discoloration. On the head are six swellings, varying in size from a cherry to a small walnut. They are fixed to the skull, and the skin over them is freely movable and not inflamed or tender. One of the largest shows a little purplish discoloration like that of a hæmatoma. Each swelling is round, well-defined, smooth, firm but not very hard. The left side of the face is a little swollen. No glandular enlargement. Since admission more swellings have appeared and there are now (May the 27th) no less than fifteen, all of them under the scalp, and most of them under the frontal portion. The earlier lumps have also increased in size. There is much purplish discoloration of the eyelids on both sides. The face is swollen, white, and waxy, mainly on the left side. There is a considerable hard swelling over the left zygoma and malar bone. The left eye is involved and somewhat proptosed. Vision is deficient and a white growth can be seen in the posterior chamber with retinal vessels running over it. The glands in both submaxillary regions are a little enlarged. No evidence

of secondary growths elsewhere can be found. The general condition of the child is deteriorating, although she remains at about the same weight.

The main interest of the case lies in the distribution of the secondary growths.

(*Exhibited May the 27th*, 1904.)

Discussion.

Mr. George Pernet said that some years ago at University College Hospital the body of a new-born child was brought with a number of sarcomatous growths in the skin. He had had an opportunity of examining these growths histologically, and they were certainly sarcomata.* But the point of special interest was that the right eyeball was very prominent and its tension greatly increased. Unfortunately, the parents would not hear of post-mortem examination. There were seventy growths scattered all over the body. Dr. Cautley's case also reminded him of another case he had seen, viz. in a woman aged about 35 years. The growths on the scalp were purple in colour, and several of them had broken down. If in the case now exhibited the sequel were fatal it would be desirable to have a histological examination made.

Postscript (*October*, 1904).—The child developed secondary growths in the groins and died about four weeks after being shown before the Society.

75. SARCOMA OF A MEDIASTINAL GLAND.

By EDMUND CAUTLEY, M.D.

Samuel B—, aged 3 years and 7 months, was admitted into the Belgrave Hospital for Children under my care on March the 29th, and died on May the 8th, 1904. He was the second of three children, the first having died at 4 months of age, the third being healthy. He had had no previous illness and had appeared to be quite well until March the 22nd. On this date he complained of pain

* Pernet, "Congenital Multiple Sarcomata of the Skin," 'Trans. Path. Soc. of London,' vol. liii, 1902, p. 360 (with short bibliography).

about the umbilicus and in the right side. The pain came on four or five times a day in attacks which lasted for about a quarter of an hour. These attacks also occurred at night and woke him up. During an attack he became blue and short of breath. The description given by the mother suggested spasm of the diaphragm. The attacks had persisted up to the time of admission. His appetite had been bad.

On admission he was noted to be a bright, well-developed boy, who looked quite well and not at all anæmic. Examination of the chest revealed dulness over the whole of the right side, especially marked up to the second rib in front and the spine of the scapula behind. Breath sounds could not be heard below the level of the spine of the scapula, or below the level of the fourth rib in front. There was no bronchial breathing. The heart's apex was felt in the fifth space, $\frac{3}{4}$ths inch outside the nipple line. The liver was greatly depressed. Its convex upper surface could be felt in the right hypochondriac and epigastric regions, and its edge as low as the level of the umbilicus. On exploring in the sixth space on the right side in the mid-axillary line some grumous, chocolate-coloured fluid was easily obtained. This yielded a brown sediment on standing of blood-corpuscles and débris. No tubercle bacilli were found.

At 5 a.m. on March the 31st patient had his first attack of pain since his admission. He became pale, blue round the lips, and almost doubled up. The attack lasted for twenty minutes. The pain was referred to the epigastrium. Next morning at eleven and the following morning at five he had similar attacks of rather shorter duration. On April the 6th it was noted that he had had no more pain. The breath sounds were absent, except at the extreme apex, and the dulness was more marked. He had slight cough but no fever. His pulse rate was 150. On the 9th he had one slight attack of pain and the cough was rather more marked. Twenty-four ounces of dark altered blood were drawn off by tapping. The blood

became somewhat brighter towards the end of the operation and continued to flow freely. The boy, however, showed no symptoms of loss of blood. Examination yielded the same results as in the specimen removed on exploration. No cells suggestive of sarcomatous cells could be found. The blood did not clot. Although so much fluid was removed no change could be detected in the position of the heart, and very little in that of the liver.

On April the 14th the rash of measles came out and lasted for four days. The attack was a mild one and hardly made the boy feel ill. On April the 21st breath sounds could still be heard at the right apex. Breathing was rather more difficult, and the boy had some pain in the lower costal region on the right side. On April the 27th the apex of the heart was found a little more displaced outwards, its action was more frequent, and a systolic murmur could be heard. The liver was more depressed and the physical signs in the chest were more marked. Two days later marked bulging of the diaphragm in the epigastric region was noted, forming a distinct prominence. Twelve more ounces of fluid, similar in character to the last, but containing some clots, were withdrawn by tapping. No improvement in the breathing or in the action of the heart resulted from the removal of the fluid.

Gradually the patient got weaker and became more short of breath. The pain, discomfort, and dyspnœa were considerable. The action of the heart became more interfered with. The appetite was impaired, yet there was no difficulty in swallowing. Death resulted from asthenia.

Post-mortem examination.—On opening the abdomen the liver was found to be pushed down below the costal margin. The diphragm on the right side was convex downwards, with a marked bulge in the epigastric region corresponding to that felt during life. On opening the chest the heart was seen to be pushed over to the left. The left lung was normal. Occupying the whole of the right side

of the thorax was a large solid mass, adherent to the diaphragm and the chest wall, except over the upper lateral and anterior portions. Here it was separated from the chest wall by a thin layer of compressed airless lung. The lung was adherent to but was not infiltrated by the tumour.

On section it appeared that the growth had started in, and spread from, one only of the posterior mediastinal glands. It was very firm, white, and solid, except in the outlying parts of more recent growth, which were soft and hæmorrhagic. A considerable mass of blood-clot was present in the peripheral portion, separating the growth from its capsule and creating a large irregular cavity into which papilliform processes of growth extended. It was from this cavity that the fluid was obtained during life. In the axillary region the cavity was merely separated from the chest wall by the capsule of the growth.

No secondary growths were found and no other abnormalities.

Microscopically, the tumour proved to be a pure round-celled sarcoma.

The diagnosis of the case was somewhat difficult at first, for the ease with which fluid was obtained, and the amount drawn off, indicated that the case was one of hæmothorax. The most probable cause, therefore, was tuberculosis of the pleura or new growth. Not until it was evident that the removal of a large amount of fluid did not affect the position of the heart could a more accurate diagnosis of the tumour in the posterior mediastinum be made.

It is noticeable that the tumour must have been in existence for a very considerable time before it gave rise to any symptoms. Probably the first symptoms were caused by involvement of the diaphragm and hæmorrhage into the tumour.

(*Exhibited May the 27th*, 1904.)

76. PARALYSIS OF THE TRAPEZIUS MUSCLE SECONDARY TO SHINGLES.

By WALTER EDMUNDS, M.C.

The patient, a little girl, aged 6 years, was brought to the Evelina Hospital Out-patient Department suffering from a herpetic rash over the right scapula. A fortnight later the rash had disappeared, but there was noticed some deformity about the shoulder, which on examination proved to be due to paralysis of the lower part of the right trapezius muscle. An electrical examination of the case was kindly made at the Royal Free Hospital. The lower part of the muscle was found not to respond to either faradism or galvanism. No other muscle appeared to be affected. Cases of paralysis of the trapezius are not very rare, but it is exceptional for one to be preceded as this was by an attack of herpes in the neighbourhood of the muscle.

(*Exhibited May the 27th*, 1904.)

Discussion.

Mr. George Pernet said he had seen paralysis of the facial nerve follow herpes zoster of the cervical nerves on the right side corresponding to the third and fourth ganglia.

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77. CASE OF ACHONDROPLASIA.

By HAROLD BALME

(Introduced),

AND

A. D. REID

(Introduced).

The case of achondroplasia is that of a boy, H. M—, aged 12 years. At the time of his birth his mother was aged forty-four, six years having elapsed since the birth

of her previous child (though she had had a miscarriage three years after that event). Altogether the mother had had nine previous pregnancies, of which the following is the history:

First child died at the age of six of "convulsions."

Second child died at the age of two, also of "convulsions."

Third child, a girl, is still alive and quite healthy.

Fourth child died at the age of seven of diphtheria.

Fifth child, a girl, is still alive and quite healthy.

Sixth child, a girl, also alive and healthy.

Seventh child, a boy now aged twenty, in good health.

Eighth child died at the age of four of measles and bronchitis.

The ninth pregnancy miscarried at the third month.

The mother states that throughout the whole length of time she was pregnant with the patient she menstruated regularly every month; also that during this time she was subject to several shocks.

The confinement was a difficult one owing to the large size of the child's head, and the fact that it was a breech presentation. No instruments were employed, but artificial respiration had to be resorted to, as the infant was in a state of partial asphyxia. As soon as he was born he was noticed to have a large head and very tiny arms and legs. Up to the age of fifteen months he was fed solely on the breast, and appears to have been quite healthy, though he is said to have had a fit when nine months old. Dentition occurred at the ordinary time, but the mother did not allow him to attempt to walk until he was a year and nine months old, as "he was so fat." He then made rapid progress, and has always been particularly strong, never suffering from any of the ordinary children's ailments with the exception of measles. He has always been very bright mentally, but was not sent to school until two years ago, as he was so much teased by other children. Since then he has done very well, and seems to possess good intelligence and a nice disposition. At the present

time (at the age of twelve) his height is only 38¾ inches, and he presents the following special features of the affection :

The head is abnormally large and square, measuring 23¾ inches in circumference, but is free from bosses, or other signs of disturbed growth. The bridge of the nose is very depressed, and the mouth is usually kept open. (The child has adenoids in abundance.)

The thorax is well developed, and measures 24 inches after deep expiration. The clavicles are normal in length, though rather more curved than usual. The scapulæ and sternum also appear normal, and there is marked beading of the ribs. The vertebral column measures 20¾ inches from the occipital protuberance to the tip of the coccyx, and there is well-marked lordosis. The thyroid body is palpable, and there are no supra-clavicular pads.

The arms are very short, so that the fingers only just reach the bottom of the great trochanter when the boy is standing upright. The humeri are short and thick and slightly curved, and the elbow-joints cannot be extended beyond an angle of about 160°. The hands are small and fat, and the fingers all of the same length; these show very clearly the characteristic separation between the middle and ring fingers.

The femora are short and thick, the lower epiphyses appearing relatively broad and massive. There is a marked lateral curvature of the left tibia and fibula, but the corresponding bones on the right side are quite straight. The feet are somewhat deformed, the middle toe of the left foot being curiously deflected laterally, so as to lie over the second toe.

The following measurements of the chief bones, etc., are given in parallel columns with those of a normal boy of the same age, for the purpose of comparison :

	Achondroplasic patient. In.	Normal boy of same age. In.
Height	38¾	55¼
Height when sitting	26	27½

	Achondroplasic patient.	Normal boy of same age.
	In.	In.
Crown of head to umbilicus	20	22½
Umbilicus to sole of feet	18¾	32¾
Supra-sternal notch to symphysis pubis	16½	15¾
Occipital protuberance to tip of coccyx	20¾	24
Circumference of head	23¾	20¾
Tip of mastoid to tip of mastoid (across vertex)	16¾	15¼
Clavicle	5⅛	5⅛
Humerus (acromion to external condyle)	5½	10¼
Radius	5	7¼
Ulna	5½	8
Wrist-joint to tip of middle finger	5¼	5¾
Circumference of hand	5¾	7
Thumb	1⅜	2
First finger	1⅝	2⅝
Second finger	1⅝	2⅞
Third finger	1⅝	2¾
Fourth finger	1⅜	1⅜
Circumference of upper end of humerus	8½	10
Circumference around crest of pelvis	21	22¼
Anterior superior spine to knee-joint	7	16
Anterior superior spine to internal malleolus	15½	28¼
Great trochanter to sole	18½	29¾
Femur (great trochanter to external condyle)	8¼	13½
Tibia	6¼	11⅜
Fibula	7¼	12½
Length of foot	6½	8¼
Circumference of lower end of femur	10	11⅜

The above measurements not only show the great diminution in size of the bones affected in this deformity, but they also confirm the fact that membrane bones are unaffected, and a comparison of the heights of the two boys

when sitting proves that the dwarfed stature of the achondroplasic patient is entirely due to the smallness of the lower extremity. A comparison is also given of the circumference of the upper end of the humerus and of the lower end of the femur in each case, in order to demonstrate that the so-called thickening of these bones is more apparent than real.

X-ray examination.

Main features.—Long bones straight. Epiphysial line sharp. Clavicle, scapula, and spine normal. Epiphyses of normal size.

Abnormal features.—Ends of diaphyses expanded both antero-posteriorly and laterally, especially the upper and lower ends of the femur, tibia, humerus, ulna, and radius. Also the anterior ends of the ribs, producing "beading." Bowing of both humeri, with concavity forwards. Lateral curve of the left tibia and fibula. Notching of the ends of the diaphyses, especially the upper ends of both humeri, the distal ends of the second metacarpal bones, and the proximal phalanx of the third left toe. Stunting of all the long bones (except clavicles). The second left metatarsal is especially affected, accounting for the deformity of the toe. The phalanges are short and broad.

(*Exhibited May the 27th*, 1904.)

Discussion.

Mr. George Pernet pointed out that achondroplasia was a very ancient disease, and in the Egyptian galleries of the British Museum there were quantities of glazed figures of which representations appeared in the first number of 'The British Journal of Children's Diseases.'* He showed a photograph of a similar figure unearthed at Carthage. † He showed also a small clay figure he had brought back recently from Carthage, and which he thought was identical, but Dr. Wallace Budge, the keeper of the Egyptian antiquities at the British Museum, was not sure of this. He (Mr. Pernet) thought the figures had been imported from Egypt, but Dr. Wallace Budge said it was not so, as the Carthaginians made them themselves. The achondroplastic Ptah-Asar-Seker represented the triune god of the resurrection,

and it seemed peculiar that the Egyptians should have thus deified a deformity. The very small clay figure which Mr. Pernet handed round was said to be the god Bes. It was very poorly made and the condition was not easy to make out. The god Bes appeared at any rate to represent a cretin.

* Pernet, "A Note on the Antiquity of Achondroplasia," *loc. cit.*, January, 1904, p. 7.

† R. P. Delattre, "Lettre sur les fouilles de la Nécropole voisine de Sainte-Monique, à Carthage," 'Acad. des Inscriptions et Belles-Lettres,' 1900, p. 83, Plate II.

78. A CASE OF BILATERAL SEPARATION OF THE LOWER EPIPHYSIS OF THE RADIUS.

By J. W. THOMSON WALKER, C.M.

The introduction of the X rays has added very materially to the accuracy of diagnosis in bone injuries, but it is probably in the examination of epiphysial lesions that this means of diagnosis has proved of greatest value.

The following case is of sufficient interest to merit record, the more so that there are few skiagraphic records of such cases. William J. W—, aged 15 years, fell from the back of a railway van on to the street, alighting on his outstretched hands.

He was conveyed to the North-West London Hospital, and there examined by the House Surgeon, Mr. E. A. Wright, who found both hands apparently dislocated backwards. Some amount of swelling had already taken place when he was examined. The carpus appeared to be dislocated on to the back of the radius, the lower end of the latter projecting on the front of the wrist. The condition was similar on both sides. Reduction was easily effected and was accompanied by indistinct crepitus. Anterior splints were applied. On removing the splints three weeks after the injury, Mr. Wright was dissatisfied with the appearance of the wrists and referred the case to me. The following condition presented itself on examination.

The hands viewed laterally showed the "silver fork deformity," but to a less degree than in Colles' fracture. There was radial inclination of both hands, which was more pronounced on the right side. The head of the ulna was prominent on both sides. A finger-breadth above the wrist line bounding the thenar eminence there was a prominent transverse ridge in the line of the radius which could be distinguished as a sharp edge covered by the wrist tendons, and was best felt by pressing the finger from below upwards along the front of the wrist. On the dorsum the carpus was prominent and was found to be bounded by an abrupt depression over the back of the radius. The position of the styloid process of the radius appeared to bear its normal antero-posterior relation to that of the ulna, but as both wrists were injured there was no standard for comparison. On measurement the tips of the radial and ulnar styloid processes were equidistant from the humeral condyles on the left side, and the right radial styloid process was a quarter of an inch higher than the ulnar styloid process. There was thus shortening of the radius on both sides, that on the right side being most marked. A skiagram taken from the inner side of each wrist showed that the epiphysis of the radius was separated and displaced backwards so that the anterior edge of the cartilaginous plate rested upon the middle of the lower end of the shaft of the bone and the posterior half of the epiphysis projected backwards beyond the shaft. A ledge of bone had been broken from the posterior edge of the lower end of the shaft and displaced backwards with the epiphysis. The lower epiphysis of the ulnar was uninjured on either side.

Under an anæsthetic the epiphyses were found to be so firmly united to the shaft in their abnormal position that it was impossible to dislodge them.

Seven weeks after the accident the inflammatory swelling around the wrists had disappeared and the deformity was less noticeable. The abduction of the hand was still more marked on the right side and the measurements were

Fig. 20.

FIG. 21.

unchanged. The ridges formed by the lower end of the shaft of the radius on the anterior aspect of the forearm and that formed by the epiphysis on the dorsum were less sharp and prominent. The movements of the wrist joints were perfect and could be carried out without difficulty or pain. In this case there had apparently been a complete backward displacement of the lower radial epiphyses at the time of the accident, and this having been reduced, the partial displacement, seen in the skiagram, remained. The rapid firm union of the cartilaginous disc in its new position is what usually occurs in such cases. It is satisfactory to learn from the experience of others that the deformity in these cases may be expected to disappear completely and leave no impairment of the movements of the wrist. This certainly appears to be taking place in this case even after the lapse of seven weeks.

The serious question of the arrest of growth of the shaft of the bone with consequent distortion of the limb must always arise in these cases. So far as I can ascertain there is no very precise knowledge of the factors which govern the arrest of growth in separation of the epiphyses. Severe comminution, destruction of the soft parts, and suppuration are naturally followed by impaired activity of the injured epiphysis, but in simple detachment of the radial epiphysis the after result varies considerably.

The radius has been stunted in growth after simple separation with little or no displacement, and in other cases, where the epiphysis has been completely displaced, the injury has not interfered with the full development of the bone.

It is, therefore, impossible to say in a case like that just described whether arrest of growth with its serious deformity may not take place.

(*Exhibited May the 27th*, 1904.)

79. SPECIMENS FROM A CASE OF STATUS LYMPHATICUS.

By HUNTER TODD, B.C.,

AND

WALTER EMERY, M.D.

(Introduced.)

The specimen was taken from a boy, aged 10 months. The patient was brought to the Paddington Green Children's Hospital in January, 1904, by his foster-mother, who stated that the child had such difficulty in breathing that she was in constant fear it would some day suddenly choke. The child was rickety. There was obvious stridor, chiefly inspiratory, worse on crying. The stridor had existed, more or less, since birth; when the child slept or was quiet it sometimes disappeared.

Examination of the larynx showed that the epiglottis was markedly folded over on itself and compressed laterally, so that a view of the interior of the larynx could not be obtained, and it was considered to be an example of the type of larynx which has been described by Drs. Sutherland and Lack as being the cause of the so-called congenital stridor. The child did not improve under treatment.

Three months later, while waiting in the Out-patient Department in order to be seen by a surgical colleague, to whom he had been sent to be treated for a rickety curvature of the spine, he began to cry. After a minute or two he appeared to be seized with laryngeal spasm. There were rapid inspiratory efforts, but no air-entry into the lungs. The house-surgeon, who was present at the onset, tried artificial respiration, which was of no avail. Tracheotomy was then performed and artificial respiration again attempted, but it was too late. The child was dead.

At the onset of the laryngeal spasm the child became cyanosed, but this was very rapidly succeeded by extreme pallor and loss of pulse.

Curiously enough, two days later another child died

during the administration of chloroform. This patient was aged 11 months, and had no sign of any laryngeal trouble.

The house-physician, who was acting as anæsthetist, noticed the breathing had ceased, whilst the abdominal movements were exaggerated. The mouth was opened, the tongue pulled forward, and the finger passed down to the larynx to discover any obstruction. It was impossible to get the finger into the larynx, as the glottis seemed to be tightly closed. Suddenly the glottis opened with a distinct "click," and the respiration was resumed for a brief moment. The breathing ceased again, and in spite of artificial respiration, the child died. He did not get cyanosed, but was pallid throughout, and the pulse could not be felt at the wrist.

In both these cases there seems at first to have been laryngeal obstruction due to spasm, followed by cardiac failure. The post-mortem examination showed no sign of death from asphyxiation, and the sudden pallor, loss of pulse, and rapidly ensuing death also pointed to cardiac failure.

In the first case the predisposing cause of the laryngeal spasm may have been the peculiar form of the larynx itself; in the second case it was probably brought about by the irritating effect of the chloroform vapour.

The point of interest in both cases was the post-mortem discovery that in both there was a considerable enlargement of the thymus gland. To what degree was the cardiac failure due to this enlargement?

(*Exhibited May the 27th*, 1904.)

Discussion.

Mr. Thomson Walker remarked that the condition under discussion had not been very fully recognised in this country, and he was glad to hear it was being looked for. When he was in Vienna he saw a number of such cases, and went through the literature of the subject. The first author to draw attention to the subject was Nordmann, a Swiss authority, who described a number of cases of sudden death in water. A man got into the water and was taken out dead before he had been immersed many seconds, and the condition now under discussion was

found. Paltauf took up the subject more generally, and showed that it was the same condition as had been looked upon as "asthma thymicum" in children. Some discussion arose as to whether death was due to pressure on the trachea. Paltauf pointed out that if it were due to pressure on the trachea there must be some post-mortem signs, in the same way as in pressure on the trachea from thyroid enlargement. He also showed that before the sudden death the persons seemed quite healthy, and that in adults there was plenty of room at the upper aperture of the chest to allow of considerably more enlargement of the thymus than was present. One also found circulatory changes in such cases. In many there was a hypoplasia of the arteries, so that the lumen was narrowed. In Paltauf's cases the ascending part of the thoracic aorta was specially affected. Sometimes the heart was small, but in many cases it was dilated, and there was some thickening of the endocardium. The genitals were noted in some cases to be small and in women the mammæ were undeveloped. Apparently the cases did not live beyond 30 years of age. There was not much known about the ætiology of the condition. The immediate cause of death was some slight shock—and these cases were apparently particularly sensitive in this respect. Paltauf described some cases in which active rachitic changes were present in the epiphyses, but he described another type in which no signs of rickets were present. Possibly the condition had some relation to the other hyperplasias of the lymphatic system, such as Hodgkin's disease. It was a very important condition, not only from the medico-legal point of view, but also from the surgical, for Kundrat had collected ten cases in which death occurred either at the commencement of chloroform administration, or during it, or immediately after the operation.

Dr. E. P. Baumann said he had had the opportunity of seeing two such cases at Great Ormond Street, one of them a baby aged 3 months, who died under chloroform, and the other in a child aged 9 months. The second case was in the hospital only two hours and was brought in a state of great respiratory embarrassment, the condition having come on twelve hours previously. When he saw the child it was blue in the face, and had great respiratory distress, uttering peculiar respiratory sobs, similar to those of an older person who was being suffocated. It was too ill to be examined thoroughly, but there were rhonchi all over the chest. Death soon ensued, and it was found that the thymus weighed 1 oz.; it lay chiefly on the left side and had pressed back the heart. Upwards it extended to the right and might quite possibly during life have pressed on the trachea, producing death by tracheal obstruction. The other post-mortem appearances resembled those described in the present cases. In the duodenum the enlarged solitary follicles gave the intestines

the appearance almost of a hob-nailed boot; there were thickened patches like those described by Dr. Emery, consisting of an aggregation of follicular tissue. Whatever might be the cause of such cases in general, it seemed to him that in the second case death might have been due to actual pressure. There was a history of a similar attack a month previously, which was stated to have been worse. In the first case the thymus weighed $1\frac{1}{4}$ oz. Here, too, there was hyperplasia of the lymphatic tissues generally.

Mr. Hunter Tod, in reply, said that although he at first thought the case was one of congenital stridor, he was willing, from what he had heard, to accept the cause of the laryngeal spasm as being in some way due to enlarged thymus. He knew there were adenoids present, but the child was feeble and rickety, and he did not think he would have been justified in operating, as the small amount of adenoids in no way seemed to be producing any symptoms of obstruction to breathing.

80. SPECIMEN OF A LARYNGEAL POLYPUS.

By E. P. BAUMANN, M.D.

Dr. Baumann showed a specimen of a polypus obstructing the larynx. The case was that of a female child, aged $3\frac{1}{2}$ years, who was admitted into the Great Ormond Street Hospital for Sick Children, for loss of voice and difficulty in breathing. A laryngeal examination was attempted, but, owing to the struggles of the child, was not successful. The condition improved considerably, and no further attempts at local examination were made. After some weeks the patient was discharged. The father appeared a few days later and reported that the child had six hours previous suddenly become blue in the face and died. An autopsy was held, with the result that a polypus was found completely obstructing the larynx.

(*Exhibited May the 27th*, 1904.)

81. A CASE OF CONGENITAL DILATATION OF THE COLON.

By GEORGE CARPENTER, M.D.

J. P—, aged 5½ years, was admitted into the North-Eastern Hospital for Children on April the 25th, 1904. He had been constipated from birth, and required aperients from the very earliest days. His belly was noticed to be large during the first year of life. He was seen by me at the Evelina Hospital when he was nearly 2 years old, and subsequently he came under Dr. Goodhart's care, but no positive diagnosis, from what his parents tell me, appears to have been made by either of us.

Now, his bowels are "never opened without aperients," and opening medicines have been administered twice daily. Since November last he has been given "strong cascara," and before this Epsom salts. After passing flatus during an action he is much better, and "the measurement goes down inches," but sometimes this does not happen for a fortnight. Of late his motions have "bubbled like soda-water."

He has recently been taken to Mr. Hurry Fenwick, who diagnosed a "kink" and "advised an operation." A fortnight or so before admission he saw Mr. Barker, "who offered to take him into University College Hospital with a view to operation."

A few days before he was admitted into the North-Eastern Hospital he was sent to me by Dr. John Spurway, and I found him with an abdomen measuring 28 inches—larger even than it is at the present date, May the 6th.

He is an only child. There is no tuberculous family history. He has had measles.

On examination he is a thin boy, with rather rosy cheeks and red lips, and a very large abdomen. At times he is peevish and irritable, but since his stay in the hospital he has been good-tempered and tractable. His

abdomen is much enlarged and tympanitic and measures 27 inches in circumference.*

On inspection, vermicular movements of the intestines, which are distended with gas, are plainly to be seen. There is no definite direction in which these movements take place, nor is there any fixed spot where they cease. On auscultation of the abdomen the bell sound can be readily obtained over the situation of the ascending, transverse, and descending colon with a neutral area around the umbilicus.

On examination *per rectum* nothing abnormal can be detected there, nor is any abnormality to be found in the abdominal cavity by bimanual manipulations, apart from distended viscera.

The *cardiac impulse* is in the third left interspace in the nipple line.

The *breath sounds* are vesicular, but the intestinal tympanitic note reaches nearly to the angles of the scapulæ, and the liver and spleen areas of dulness are much encroached upon. Anything more than the lightest percussion over the liver brings forth a tympanitic note.

The *urine* is free from sugar and albumin.

The day following his admission he was given two enemata, but without result. On April the 27th the girth had diminished and was then 22½ inches. He had passed neither motion nor flatus. A rectal examination was made, and fæces were found to be present in the rectum, but the administration of an enema was not followed by a motion. On May the 2nd the abdomen measured 25½ inches, and on the 5th it was 24½ inches. Since his admission on April the 25th he had taken his meals well, but had passed neither motion nor flatus.

His temperature, which, with one or two trifling exceptions, had been normal, rose to 102·8°F, his left tonsil was enlarged, but he was apparently well and complained of

* The photograph of Dr. C. N. Gwynne's case in Vol. II of the 'Reports,' p. 130, Fig. 16, is very similar in appearance to my own case, and so much so as to make the reproduction of a photograph unnecessary.

no pain; the abdominal measurement was 24½ inches, and there had been no evacuation. The temperature fell the next day to 99° F., but it was up again on the 7th to 102° F. The respirations were 56 to the minute, the pulse 136 to the minute; the abdomen was tympanitic and the breath offensive. On May the 8th he passed a small, offensive, watery, light brown motion and flatus after a dose of castor-oil the preceding night. He looked ill and he was feverish, but there were no physical signs to account for his symptoms. His abdomen was distended, the tongue foul, and the breath offensive. A rectal tube was passed but there was no flatus. There were no Koplik's spots. In the afternoon he became delirious and was sick three times. The bowels were opened naturally immediately following the passage of a rectal tube. The following day, in the morning, a measles rash appeared, together with sneezing, and the temperature, which had been 101·6° F., fell on its advent to 99° F. There were now some Koplik's spots. On the 10th he was covered with a rash, and Koplik's spots were present in abundance; the temperature was then 102° F. The abdomen was more distended, and measured 30 inches. The tympanitic note extended on the left side to the upper border of the fifth rib in the nipple line, and to the lower border of the sixth rib in the mid-axillary line. Behind the percussion note was tympanitic as high as the angle of the scapula. On the right side the liver dulness, which was not absolute and hard to define, was in normal relationship with the ribs, and behind the note was tympanitic to within 2½ inches below the angle of the scapula. All the interspaces were increased in width, and the ribs were pushed up.

May the 12th. Still an abundant rash present, which was fading from the face, but pronounced on the trunk and legs. His belly was more distended, and measured 30¼th inches, and he had passed neither motion nor flatus. His appetite was poor, and he was sick twice the day before. A few bronchitic signs were heard. Following a turpentine enema he had a large motion of cement-like

consistency, and his abdomen decreased 1 inch in circumference. The temperature was about 99° F.

May the 14th. He was very restless, and cried on account of pain at the umbilicus. The abdomen was very tense, larger than ever, and measured 31¾ inches, and the skin over it was shiny. The heart's apex was found to be pushed up a space higher than on the last occasion when its position was noted, viz. to the third interspace. He was sick once during the night.

On the 15th he was given a turpentine enema, which was followed by a large, light, loose motion, a little flatus, and by great relief. The abdominal measurement decreased 2¾ inches. The temperature was not quite normal; it reached 101° F. on this date, as also on the 14th.

On the 16th another enema was given him, which was followed by a large motion and a decrease in size of an inch.

On the 17th the temperature was 99° F. He was given another turpentine enema, which was followed by a good action.

On the 18th Mr. Douglas Drew performed an inguinal colotomy. On performing the operation the peristaltic contraction was very strong, and made stitching difficult. The gut was, however, brought into the wound and stitched to the abdominal wall; it was not opened. There was a considerable quantity of ascitic fluid in the abdomen at the operation. He slept well during the night.

On the evening of the 18th the pulse was very rapid. He passed two stools naturally, one of them loose and large, the second formed and large.

The bowels were opened three times on the 19th, the motions being very loose. He was covered with a punctate erythematous rash. His tongue was very coated, and the mouth was foul. He was very restless, and constantly called out for drink.

He had two motions on the 20th, on which date the rash was still marked, and one on the 21st. On the last date some tubular breathing was heard about the angle of

the right scapula. The abdomen was not nearly so distended. The rash was fading. The temperature had risen since the operation, the highest being 103·9° F., the lowest 99° F.

May the 22nd. He passed a very restless night, vomited twice, and complained of slight abdominal pain. His face had a pinched expression, and his general condition gave cause for anxiety. Nothing fresh was detected in the chest. The rash was still present. The wound was not looking healthy, and adhesions had apparently not formed. He died quite suddenly that night.

The post-mortem examination was made by Mr. Douglas Drew. On inspecting the wound all the stitches had cut out, and there were signs of peritonitis. On opening the abdomen, acute peritonitis was seen to be present. In the right flank there was a large coil of distended gut, presumably the ascending colon: running diagonally from the right iliac region to the left hypochondrium was another enormously dilated coil of large intestine. In the left iliac region there was a large coil of the sigmoid, part of which had been stitched into the wound at the operation. The intervening space between the two last mentioned coils was occupied by small intestine. On taking hold of the large diagonal coil of large intestine and turning it up, it seemed to make an abrupt turn in the right flank, and was continued into the distended coil first mentioned, and at the junction of the two the gut was somewhat contracted.

On tracing the coil, which occupied the right flank upwards to the liver, it made an abrupt turn at the hepatic flexure, and descended to the cæcum. The cæcum and ascending colon were not distended to the same degree.

On tracing the left end of the transverse colon which formed the diagonal coil, it passed to the splenic flexure, and from thence onwards into the sigmoid.

The whole of the large gut was enormously hypertrophied and distended, but the thickness of the muscular wall seemed to increase as one descended.

On tracing the enormous sigmoid into the pelvis, its size was about the same until its passage through the pelvic floor.

There was absolutely no evidence of any narrowing of or anything pressing on the gut. There was no evidence of old peritonitis in any part of the abdomen. The mesenteric glands were enlarged and hard. The small gut appeared somewhat enlarged. The stomach was enormously distended. The diaphragm on the left side reached to the middle of the second interspace—on the right side to the third rib and space. The intercostal angle was widened. The spleen, liver, and kidneys were normal. There was some fluid in the pericardium, but none in the pleuræ. There was a patch of collapse in either lung base, especially the right base. There was no stricture of the rectum.

A subsequent examination of the intestines, at which Dr. Sydney Curl assisted and made the microscopical specimens, brought to light the following additional observations. The whole mucous membrane of the large bowel was of a chocolate colour, stretching from the colon side of the ileo-cæcal valve to the anus. This, on microscopical examination, was found to be due to blood pigment deposited in the mucous membrane. There was no ulceration of the mucous membrane, but it was thought to be thickened somewhat. There was enormous hypertrophy of the circular and longitudinal muscular fibres. This was not so marked in the muscularis mucosæ, but it was very obvious. The circumference of the bowel near the cæcum was 5 inches, and at the lower part of the colon 7½ inches. The small intestine was considerably dilated; its circumference was 5⅜ths inches. Its muscular walls (the circular fibres) appeared to be slightly thickened.

The duodenum was not so dilated as the rest of the small intestine—its circumference was 3¼th inches. The stomach was very large; it measured from the pylorus to the most distant extremity of the fundus 9 inches. The vertical measurement from the cardiac orifice to the

greater curvature was 5¼ inches. Its muscular walls were thickened (the circular fibres).

All these measurements were made on the organs after they had been in a 20 per cent. solution of formalin for a fortnight. Allowance must therefore be made for considerable shrinkage.

The muscular walls of the colon in the hardened specimens were about ⅛th inch thick and of the sphincter ani about $\frac{1}{16}$ths inch. The circular fibres were a shade larger than the longitudinal. In Valagussa's case the transverse layer was uniformly thickened and the longitudinal layer thinned. As in my case, there was increased vascularity of the submucous tissue. There was neither ulceration nor cicatrices. The mucous membrane of the colon showed absence of superficial epithelium and in this respect was like Valagussa's case. The same condition was also noticed in the small intestine and the stomach. In the duodenum the absent epithelium was replaced by fibrosed cellular tissue about as thick as the circular muscular coat. The denudation of epithelium was possibly a post-mortem change.

Remarks.

The interest of the case from an ætiological point of view lies in the pathological findings. It does not appear, nor was there a shred of evidence to prove, that the dilatation of the large bowel was secondary to obstruction, and therefore that the hypertrophy was compensatory in origin. On the other hand, not only was the colon found to be at fault, but other sections of the gastro-intestinal tract were affected. Thus the small intestine and the stomach were enlarged and their muscular walls were somewhat hypertrophied, though certainly not to the proportionate extent that was noticed in the large bowel. It is a matter for regret that the œsophagus was not removed for examination, as it likewise might have shown some anatomical changes. The case suggests that the condition of megacolon is a vice of development, a con-

genital malformation, and is not an illustration of obstruction followed by over-distension and compensatory hypertrophy. It would certainly appear to be so in this instance. Certain other points strike one. Thus the absence of anæmia where one would expect its production from auto-intoxication by the bowel causes surprise. Possibly auto-intoxication was absent, which would also explain the apyrexia and the freedom from nervous symptoms which was a feature in his case while under observation. The case was quite a contrast in this respect to C. N. Gwynne's in a child aged 6½ years reported in Vol. II, p. 129, of the 'Reports,' who displayed mental hebetude and lay like a log in the bed, and also that by F. Valagussa ('Riv. di Clin. Ped.,' December, 1903), where the boy, a child aged 3 years, was comatose and suffered from nocturnal convulsions. The great encroachment on the chest capacity by the distended abdominal viscera, with the varying pressures induced by them on the heart and lungs, readily detected by physical examination and verified at the autopsy, was unaccompanied by any symptoms referable to these organs while under normal conditions. The cause for the uniform distribution of altered blood pigment found almost entirely in the cells of the mucous membrane, though a certain quantity was free, was apparently owing to mechanical congestion. The blood-vessels of the submucous coat were considerably dilated and filled with blood.

The enormous abdominal distension and the obstinate constipation, dating from birth, found in this case in no way differs in these respects from those cases which have already been placed on record.

(*Exhibited May the 27th* 1904.)

82. A CASE OF TRAUMATIC STRICTURE OF THE URETHRA IN AN INFANT.

By J. P. LOCKHART MUMMERY, B.C.

The patient was a healthy infant, aged 2 years. The mother said there was always great difficulty in getting him to pass his water; he screamed all the time, and the urine only dribbled away instead of coming in a proper stream.

Eight months previously circumcision had been performed, and the mother dated the difficulty in passing water from that time. She said that ever since the operation he seemed to have pain when he passed water, and that the water only came after considerable straining; this difficulty with the water had increased very much during the last few months.

On examination I found a tight fibrous stricture of the meatus which would not admit an ordinary probe. From the appearance of the meatus there seemed to be no doubt that when the circumcision had been done, the tip of the glans penis just around the meatal orifice had been cut away. It seemed as if a complete ring of tissue had been removed round the meatus. The urethra was practically closed up, only a minute opening being left through which the urine dribbled away after a good deal of straining.

I dilated the stricture by stretching it with a very fine pair of sinus forceps. This for a time relieved the condition, and the child was able to pass water easily, but ten days later he was brought back with the stricture as tight as ever, and with acute retention, not having passed any urine for eighteen hours, and having his bladder up to the umbilicus. The retention was relieved by giving him a hot bath, and the stricture was again stretched. After this a small piece of a No. 3 urethral bougie was obtained,

and the mother was told to pass this just into the urethra daily so as to keep the stricture dilated. This treatment seems to be effectual in keeping the urethra patent, but I am afraid that unless considerable care is taken in keeping up the dilation for some time, the child is almost certain to have trouble later on in life.

Cutting off the end of the glans in performing circumcision is an accident which is mentioned in most books on operating and minor surgery, but is, I should hope, a rare one. (*Read May the 27th*, 1904).

83. TUBERCULOUS DISEASE OF THE ELBOW-JOINT, WITH DISLOCATION OF THE UPPER END OF THE RADIUS.

By J. W. THOMSON WALKER, C.M.

J. H—, aged 15½ months. Attended the North-West London Hospital on account of pain and swelling of the left elbow. There was a history of a fall six months before, but it was only three months later that the elbow became painful and began to swell.

She was taken to a general hospital, where a diagnosis of fracture was made, and the arm put in splints for three weeks. The splints were then removed, and passive movement and massage employed. Under this treatment the elbow steadily got worse, and when I saw it the upper part of the forearm and lower arm were swollen, red, and very tender. Thickening of the upper ends of the radius and ulna and the lower end of the humerus could be detected. No dislocation could be found. There was some fluid in the joint. The fifth metacarpal on the left side was thickened.

The child had previously been quite healthy. Five miscarriages had preceded her birth, but two older children were healthy, and the father was said to be healthy.

It was evident that there was fairly extensive disease of the joint, but two elements of doubt arose. Firstly, whether a fracture had ever been present, and secondly, whether syphilis might not be a factor in the case.

The child was put on mercury, and the elbow put at rest on a gutter splint, with a mercurial ointment dressing over the joint.

In four months the condition had almost entirely cleared up, but the splint was still retained.

Unfortunately the mother neglected to attend at hospital for some months, and on her reappearance the joint was found to be much worse. Flexion and external rotation were especially limited, and the upper end of the radius could be felt projecting forward.

A skiagram taken from the outer side showed a dislocation forward of the upper end of the radius.

Recently a small collection of tuberculous material ruptured on the inner side of the joint, but by probing I could not detect bare bone.

There was no history of injury immediately preceding the detection of the dislocation.

The case was one of tuberculous disease of the elbow-joint, and was interesting on account of the difficulty in diagnosis in the earlier stages and the spontaneous appearance of the dislocation.

(*Exhibited May the 27th*, 1904.)

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84. A CASE OF TUBERCULOUS PERIOSTITIS.

By LOCKHART MUMMERY, B.C.

The child is 15 months old. Between two and three months ago the mother noticed that the right cheek was swollen; she says that the swelling was hard and not particularly tender. Soon afterwards she observed that

the right hand was not used, and that the middle finger of that hand was swollen and painful to the touch.

A month before coming to the hospital a discharge from the left ear was discovered.

The child has never been very strong. There is no history or evidence of syphilis, and no definite history of tuberculosis in the family.

It was breast fed, and had not been weaned when it was first brought to the hospital.

The patient was first seen on April 14th. It was pale and sickly looking. There was a large fluctuating swelling on the middle finger of the right hand.

There was also a hard, indurated swelling over the lower part of the right radius. In the substance of the right cheek there was a firm, indurated, inflammatory swelling, apparently situated in the cellular tissue. There was a perforation of the left tympanum, and considerable purulent discharge from the middle ear. The throat and fauces were normal, and there was no history of measles or other infectious disease.

Treatment has so far been chiefly directed towards feeding up the child and insisting on its getting out in the fresh air for a large part of each day. It has had cod-liver oil and hypophosphites. The ear has been treated by insufflation with boracic acid powder.

(*Exhibited May the 27th*, 1904.)

85. SARCOMA OF THE KIDNEY.

By FRANCIS JAFFREY.

Mr. Francis Jaffrey showed a girl aged 6½th years, with a tumour occupying the left half of the abdomen.

With the exception of some of the ordinary infantile complaints she had not had any previous illness. Her

father and mother were alive and well. There was no history of malignant disease in the family, but there was, however, a history of consumption on the mother's side.

Two months ago she passed blood in the urine; this lasted for nearly a week; at the time the child complained of pain on the left side of the abdomen, and she had some difficulty of micturition. These symptoms passed off, but on May the 10th she complained of pain in the left leg, and on May the 15th the mother noticed a swelling in the abdomen. She was admitted into the Belgrave Hospital for Children on May the 16th.

On admission.—She is a bright, intelligent, and well-developed child, and does not complain of any pain in the abdomen.

The tumour practically fills the left side of the abdomen: its right margin extends somewhat beyond the middle line. It is smooth and elastic and does not fluctuate, and is fairly freely movable. The urine is acid; sp. gr. 1025; no blood; no albumin; no sugar; nothing abnormal microscopically. The quantity of urine passed daily is small, viz. 14 oz. She has not lost weight since admission.

Mr. Jaffrey was of opinion that it was a case of sarcoma of the left kidney, and he asked the opinion of members as to the possibility of removing it successfully.

(*Exhibited May the 27th*, 1904.)

The Chairman (Mr. Walter Edmunds) thought the question turned upon the after-history of the successful cases of the kind. The operation could be attempted and then stopped if the kidney were found to be adhering to surrounding parts. He would like to know if Mr. Jaffrey had looked up the literature of the after-history of such cases.

Mr. Thomson Walker said that an increasing number of renal tumours were being removed from children with immediate good results and with a good after-history—*i.e.* of three or five years in which no recurrence had taken place. Abbey, of New York, had had two such cases; Israel, of Berlin, had had one; and Malcolm, of London, another. Each case must be considered on its own merits, and if the tumour were examined from within the abdomen there would be a better chance of ascertaining whether it was removable, and whether there was likely to be a recurrence.

Operations on large tumours of the kidney, he thought, must always be intra-abdominal. Large tumours of the kidney could not be removed extra-peritoneally, and there was therefore no disadvantage in first opening the peritoneum for exploration. Laparotomy should be done and the tumour carefully examined from the front, and the presence of adhesions or secondary deposits ascertained. He had not seen the present case, so could not speak definitely about it.

Mr. Douglas Drew said the point which would decide as to operation in his mind was whether there was extension of the growth into the surrounding structures, assuming it to be sarcoma of the kidney. Sometimes it was not easy to diagnose between retro-peritoneal sarcoma and one arising in the kidney itself. He had had experience of only one case, and that was fatal after removal from shock.

Mr. Watson Cheyne thought the tumour was probably malignant, but that the child would live longer if it were left alone, as the tumour probably involved the kidney pedicle. He did not think it was likely to be got away clean, and the shock might kill the child.

Mr. Clement Lucas agreed that it was probably a tumour of the kidney, but did not quite agree with Mr. Cheyne on the question of operation; because if it were left alone it would be absolutely hopeless, whereas if it were operated upon, although the statistics of such cases were extremely grave, there would be afforded a chance for the child if care were taken that it did not die of collapse during the operation. The first successful case done in this country was by Mr. Jessop, of Leeds. He had not examined the case carefully, but thought operation was not necessarily excluded.

86. A CASE OF PATENT DUCTUS ARTERIOSUS.

By E. CECIL WILLIAMS, M.B.

This case was first seen more than two and a half years ago, when a loud and prolonged murmur of roaring character, continuous throughout the whole cardiac cycle, but loudest during systole, was heard in the second left intercostal space. It was seen by some of my colleagues, who thought it might be a patent ductus arteriosus, or

pulmonary stenosis. The only symptoms were: some shortness of breath on exertion—no cyanosis—heart slightly enlarged upwards, and apex beat just outside the nipple line.

The peculiar character of the murmur has now disappeared, only a faint systolic bruit being audible. To-day the murmur is more audible, but not of the same intensity as two and a half years ago.

It seems probable that the diagnosis of patent ductus arteriosus was the correct one, and that gradual occlusion has been taking place.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th*, 1904.)

Discussion.

Dr. Roxburgh (Weston-Super-Mare) mentioned that Dr. Williams stated there was no cyanosis, but he (Dr. Roxburgh) thought there was a slight bluish tint present. He regarded cyanosis as a strong point in favour of patent ductus arteriosus. And he did not see how patent ductus arteriosus could exist without a certain measure of cyanosis.

Dr. Frederick Taylor said that he had been unable to see the case, and would therefore like to know what were the so-called sounds of the heart—whether any sounds as such were audible, or whether they were entirely drowned or absent. Also, did the murmur begin at the commencement of what might be considered the systole, or later? Was there any kind of pause? He rose because the subject of patent ductus arteriosus came before the Clinical Society only a few months ago, and a case was exhibited which had the characters which had been attributed to patent ductus arteriosus by some former observers. The patient was a young woman of between twenty and thirty. There was no cyanosis, and the murmur was a long one, apparently covering both systole and diastole. A fortnight after the meeting of the Clinical Society he, Dr. Taylor, saw at St. Bartholomew's Hospital two cases which were very much alike, and both presented similar characteristics. There was a very short sound first and then a very long murmur, having a wavy character. A similar long sound had been noticed in cases which subsequently proved to be instances of patent ductus arteriosus. He saw one a long time ago which was reported by the late Dr. Hilton Fagge, and he had seen a few similar cases since. All the cases he had seen and read of had been in adults, and cyanosis had always

been a minor feature if it was present at all. He did not think there was any necessity for cyanosis, as he supposed the blood went from the aorta into the pulmonary artery, and there was not necessarily any obstruction of the circulation. If there was an obstruction of the pulmonary artery, which was perhaps the only other diagnosis which one could make of a simple case, he supposed that cyanosis was almost necessary. Perhaps these cases get some cyanosis later in life, but the cases he had seen were in the early stage of cardiac difficulty, and that had been benefited at least temporarily by moderate rest and cardiac tonics.

The Chairman (**Dr. Theodore Fisher** [Bristol]) said the points of interest in the case were, first of all, the continuous character of the murmur, which was present both during systole and diastole, and secondly, the fact that it was very much less loud than before. He would be glad to hear whether members thought the murmur was diagnostic of patent ductus arteriosus, or whether it was thought to be a pulmonary stenosis. Similar murmurs had been noticed by observers to disappear. He said there was also a case at the hospital almost precisely similar, only the murmur was louder, in a woman aged 29 years. There was a very loud sound, which probably would be called the second sound, in the middle of the continuous murmur. It was with little doubt the second sound, because it occurred at the end of the more forcible part of the murmur, which came up to it and waned away again afterwards. A point which would possibly be in favour of the condition being patent ductus arteriosus was, that a similar murmur could be heard in association with aneurysm when it opened into the pulmonary artery or into the vena cava. Dr. Michell Clarke had had one or two such cases under his care, which he—Dr. Fisher—had reported, and also had the opportunity of making a post mortem.

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87. A CASE OF CHRONIC GRAVES' DISEASE.

By E. CECIL WILLIAMS, M.B.

About eight months ago the mother noticed the child, a girl of twelve years, was not well, and her eyes seemed more prominent. When seen five months ago at the Children's Hospital, she complained of shortness of breath on exertion. There was some prominence of the eyes.

Graefe's sign not present. There was no enlargement of the thyroid gland. Pulse 160 to the minute. Slight muscular tremor. Care should always be exercised when recommending even slight operations in these patients, as it is possible to convert them into acute cases, with fatal results. A case bearing on this point was reported by me in the 'London Hospital Gazette,' May, 1903.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th*, 1904.)

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88. NOTES OF A CASE OF CONGENITAL STRICTURE OF THE ŒSOPHAGUS.

By N. H. BERTRAM ROGERS, M.D.

Cases of stricture of the œsophagus are so rare that I have thought it worth while to bring this one before the Society.

Wm. N—, aged 1 year and 10 months, was sent from Cornwall to the Children's Hospital on April the 1st, 1904. He was a prematurely born child, and was nursed by his mother for three weeks only, after that being fed on milk and patent foods. For the first ten months of his life he was fairly healthy, took his food well, and was only sick a few times, not sufficiently to make his mother anxious about him. But a few weeks before he came under my care he had been frequently sick, bringing up his food unchanged soon after a meal, and mixed with a large quantity of mucus. So severe had the vomiting been that the child had lost weight considerably and the medical man under whose care he was had resorted to rectal feeding. When admitted, the first thing that struck me was the extreme emaciation of the child; he weighed only 16 lbs. 9 oz. He was rickety, though not excessively so, and was constantly crying—I believe, from hunger. On examination, the veins in the neck were observed to be

very full, swelling up considerably when the child cried, and filling from below. The neck was thin but the lower part seemed full, though no definite enlargement of any organ could be distinctly made out. The lungs were full of mucous rales, and whenever he vomited a large quantity of mucus was expelled. No dulness could be found in the thorax. The temperature was subnormal. Vomiting came on after every meal, and milk given him was returned, as a rule, uncurdled and neutral to test-paper. To test how long he retained food we fed him every four hours with alternate coloured and uncoloured meals, using methylene blue, fuchsine and chocolate, also having the vomit tested for acidity. We found after several days' observation that most of the food went into the stomach and was absorbed, but he would retain somewhere, either in an œsophageal pouch or in the œsophagus, food for as long as eight hours before returning it. Sometimes the vomit came from the stomach.

The passage of a tube did not give us much help. Several times it was impossible to get beyond a point just behind the upper part of the sternum, on other occasions the tube went into the stomach and returned with an acid fluid on the tip, while often it seemed to hitch near the cardiac end of the œsophagus and coil up on slight pressure. In fact, the passage of the tube helped us very little.

I also tried if I could see a pouch by the X rays, and had the child fed with a biscuit mixed with bismuth carbonate, but got no result.

The child lost weight rapidly and was evidently doing very badly, but I was surprised when on April the 13th the bronchial condition became much worse, there was much stridor, and finally he became cyanosed and died on the 14th.

The specimen which is before you shows that there is a fairly tight stricture about an inch from the lower end of the œsophagus. How it arose is impossible to say, but perhaps the same conditions that produce the pyloric

stricture of children may have caused this. None of the causes put forward appear to me to satisfactorily explain why either develop.

The diagnosis lay between a pouch and a stricture, and I was inclined to the former. The former is rare, a stricture almost, if not quite, unrecorded.

(*Read at the Provincial Meeting, Bristol, June the* 18*th*, 1904.)

89. DUODENAL ULCER IN A CHILD FORTY-FOUR HOURS OLD.

By H. W. KENDALL.

(Introduced.)

The child was born quite healthy and of good weight. Twenty-seven hours after birth it vomited a quantity of blood, and at the same time passed blood *per anum*. It passed a little more blood a few hours afterwards, and was very weak. Thirty-six hours after the first vomiting it passed more blood, and died from hæmorrhage. He would like to hear views as to the cause of the ulcer.

The mother was very healthy, but he did not know anything about the father. The child was illegitimate. Iced milk was given in order to stop the hæmorrhage if possible.

(*Read at the Provincial Meeting, Bristol, June the* 18*th*, 1904.)

90. CASES OF CONGENITAL DEFORMITIES.

By H. ELWIN HARRIS, M.B.

(Introduced.)

Case 1. *Congenital absence of the left ear with facial paralysis.*—In this case there is no trace whatever of an ear upon the left side: the only change from the smooth skin in that region is a small papule, cartilaginous to the touch, with a small depression in its centre. There is and has been from birth well-marked facial paralysis.

The soft palate is distorted, the affected side being broader and higher than the other side.

Case 2. *Congenital absence of the right external ear, excepting a partial development of the auricle.*—There is apparently some sense of hearing on that side.

In the first two or three months of her pregnancy the mother was visited by a minister who was affected in the same way.

Case 3. *Intra-uterine amputation of three extremities; webbed fingers on the hand of the only limb.*—Both legs are absent from 1½ inches below the hip-joint, and the left arm is removed flush with the shoulder-joint. The fingers of the remaining hand are webbed together to their tips. On the right stump is a fanciful resemblance to a very minute leg. One could trace the knee-joint, ankle, and an attempt at a foot. He did not think any satisfactory explanation had been given of those deformities. The idea of amniotic adhesions was, he thought, insufficient to explain them.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th*, 1904.)

Discussion.

Mr. R. Clement Lucas said he thought the question of deformities found at birth still required a great deal of elucida-

tion. All that he could say would be to negative the theories which had been put forward. He did not believe in the influence of maternal mental impression on the child, and he did not believe in amputation by the funis. How Mr. Harris collected such a series of cases of deformities he did not know, but it was a curious law of chance; if one met with a case of absent ear it was very likely another would be met with very soon. But taking the theory of amputation by the funis, was it likely that the funis, having amputated one limb at the thigh, close to the buttock, could proceed to amputate the other thigh close to the buttock; and not satisfied with that, that it should go on and amputate an arm at the shoulder-joint? If it were a matter of tension of the funis, having amputated one it would so relax its hold that the others would escape. But supposing it possible that the funis did amputate all those three, there was another distinct defect in that child which no funis could account for, and that was the adherent fingers on the remaining hand. He doubted if one could in any way induce the funis to so act on that hand as to produce webb fingers. He did not believe in funis amputation at all. In one of the cases of absent ear there was a history that at some period of her pregnancy the mother saw a dissenting minister who also was without one ear. It seemed a very strong point, but the rules of chance accounted for it, he thought. He did not believe in mental impression having the slighest effect on the embryo. The explanation was hunted up after birth to explain away the deformity. How many thousands of mental impressions have occurred without any resulting deformity? He thought it would eventually be shown that the real cause in these cases was some nervous defect. There would probably be found to be some nerve defect which might become hereditary, like Brown-Sequard's guinea-pigs, in which hereditary epilepsy and hereditary weakness of limbs were produced artificially. Other deformities seemed to commence too early for the nervous theory to hold. He did not know much about the artificial production of deformities, but he was told in connection with incubated eggs that if the temperature was carried too high there were likely to be altered limbs and defective development in them. It was necessary to wait for a further development of the phenomena, but he thought the nervous system would be found to account for a certain number of them.

Mr. A. H. Tubby said he largely shared the scepticism on the subject which Mr. Lucas had expressed. No one had seen a funis at work amputating a limb, but he had seen a funis wound round the body, though he was not aware of a case where the body had been divided by it. Still, portions of feet and of legs had been delivered separately, and such children had merely the stumps of limbs. Whether the mutilation was due

to the action of the funis or to the amnion or amniotic bands he did not know. No one seemed to believe in maternal impressions, and he did not know where else one must look for the causes of intra-uterine amputations, unless they were due to some bands such as the funis or amnion.

Mr. George Pernet expressed his entire agreement with the remarks just made by Mr. Lucas. It was high time the medical profession made a stand against the maternal impressions idea, a belief in which still seemed to linger in the minds of some medical men. Mr. Pernet thought that at a period so far removed from mediæval times that sort of superstition should be thrown overboard. It would be interesting to know if the mother of the second case with absence of one ear had also seen a minister without an ear. Such superstitions should not be tolerated. From the time he was a student the idea of intra-uterine amputations had been accepted by many people, but he had never been able to satisfy himself that such amputations really did take place. Such conditions were to be regarded as developmental anomalies. He could not say, of course, what the cause was, whether toxæmic or otherwise. He was glad Mr. Lucas had drawn attention to the matter in the way he had. To call such a case as had been shown congenital amputations was in Mr. Pernet's opinion erroneous.

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91. A CASE OF CURED HYDROCEPHALUS.

By J. MICHELL CLARKE, M.D.

(Introduced.)

Dr. Michell Clarke showed a case of hydrocephalus in which recovery had occurred. The case came under observation at the age of eight months, and the first symptoms probably appeared at about six months. The condition in the following three or four years was very marked, the child being unable to sit up or stand. Intelligence was always very good. After this it slowly began to recover power in the limbs, and the head ceased to increase in size. Improvement had gone so far that twelve months ago suitable iron supports were fitted to the legs, and since then there has been a steady recovery of power. There are signs of partial degeneration of the

pyramidal tracts, in rigidity, and exaggeration of reflexes. The head measures now nearly 23 inches in circumference. The treatment was by small doses of Hydrargyrum c. Creta, continued over long periods. The case was shown on account of the rarity of recovery from such a condition.

(*Read at the Provincial Meeting, Bristol, June the* 18*th*, 1904.)

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92. INTERNAL DRAINAGE OF THE VENTRICLES IN A CASE OF HYDROCEPHALUS.

By C. A. MORTON.

As it seems desirable to record any cases in which a new method of treatment is adopted, I have thought it well to bring this one to your notice this afternoon. The method of operating for hydrocephalus employed was suggested by Mr. Watson Cheyne and Dr. Sutherland,* and has been employed by Mr. Watson Cheyne and other surgeons. I may perhaps remind those of you who have read Mr. Watson Cheyne and Dr. Sutherland's paper that the object of the operation is to establish a communication between the distended ventricle and the subdural space, so that the fluid may be readily absorbed by the meningeal veins. I think the record of my case proves that such a communication may exist for some considerable time, and yet the hydrocephalus may increase.

The baby, K. H—, aged 7 months, was under my care in the Children's Hospital in 1900. She was born with hydrocephalus, and the head had been enlarging out of proportion to the growth of the infant ever since birth. Between August the 25th and December the 19th it had increased 1¼ inches. There was constant nystagmus, and alternating internal strabismus. The limbs were not paralysed. The enlarged head was tense, and all the bones

* 'Brit. Med. Journ.,' October the 15th, 1898.

were separate. The circumference of the head was 22 inches. I operated on December the 19th. A flap ⅝ths inch wide at its base was turned down, over the extension of the anterior fontanelle downwards between the right parietal and frontal bones, and the thin membranous skull and dura mater reflected. The brain bulged out as soon as the cranial cavity was opened, and was so tightly pressed against the skull that it was impossible to get any drain between the two, without great risk of passing it into the cerebral substance. I grasped the end of a piece of fine rubber tubing two inches long with Sims' forceps, and pushed them through the cerebral cortex (which was very thin) into the ventricle. After a little fluid had escaped through the tube, and the intra-ventricular pressure thus reduced, I easily inserted the other half of the tube between the dura mater and the cortex. The tube was secured to the dura mater by a fine silk stitch, and the flap of dura mater and membranous skull was then sewn back in place by fine silk sutures. This was done quickly, as until the last stitch was put in, the cerebro-spinal fluid flowed slowly away. It came through the tube and out by the side of it. The baby was wrapped in wool during the operation, and there was no shock. As the result of the leaking of fluid during the operation, his head became quite soft, and the edges of the cranial bones prominent, with a tendency to override. There was some leaking of cerebro-spinal fluid into the dressing during the first twenty-four hours. The wound was healed and the stitches removed on the eleventh day after the operation. The infant up to this date was kept lying on the side of the head opposite to the wound. There were signs at this time that the head was enlarging again, and after this the head steadily increased in size, and six weeks after the operation it measured 1½ inches more than it did before the operation. This increase was slightly greater on the side not operated on. A month later it had increased another half inch. As there was some difference in the rate of progress of the disease on the two sides, and as I thought the rubber tube might have

become kinked and failed to act a short time after its insertion, I decided to repeat the operation on the other side, as Mr. Watson Cheyne did in one of his cases. Mr. Ballance very kindly sent me a specimen of the metal drainage tube he had been using for the operation, and I had a similar one made for use in this case. On March the 2nd I turned down a flap on the left side in an area corresponding to the operation on the right, and exposed the brain in the same way. I inserted one arm of the rectangular metal drainage tube into the ventricle, and then withdrew the fine wire in its lumen. No fluid would flow through it, the lumen was so fine. I therefore inserted a piece of rubber tube, as I had done on the other side, by the side of the metal one, and cerebro-spinal fluid slowly flowed away by the side of it. Both tubes were fixed by fine stitches to the dura mater, and the free ends placed in the subdural space. I thought I had stopped the leakage of the cerebro-spinal fluid by suturing the flap of dura mater and the membranous skull into place, but the leaking went on into the dressing, which had to be changed in twenty-four hours. There was no shock from the operation. During the next few days some leakage continued, and the bones were overriding. Ten days after the operation the wound was found healed except a pin-point aperture, from which cerebro-spinal fluid leaked. The temperature ran up to 105° F. the same evening and the baby died. It is interesting that the high temperature which has, I believe, been fatal, or at any rate been present with a fatal termination, very soon after the operation in some cases, came on in this case on the tenth day after operation. The cause of this pyrexia of death is not clear. It was certainly associated with a continuous leakage of cerebro-spinal fluid.

Post-mortem.—On opening the cranium before the cerebro-spinal fluid escaped from the ventricle, the brain was observed to have retracted from the skull to the extent of an inch. There was no meningitis. The rubber drainage-tube inserted at the first operation on the right

side was found fixed to the dura mater by the silk stitch; one end was adherent to the dura mater line quite patent, the other lay within the ventricle; it was not kinked, and after removal fluid could be readily syringed through it. By the side of the hole through which it entered the ventricle another opening had formed between the ventricle and the subdural space, one third of an inch in diameter. The edges were quite smooth, and the opening round. It did not present at all the appearance of a tear. From the neighbourhood of the opening a thick band of adhesion an inch in length extended to the dura mater. On the left side the brain was adherent to the scar. Both the rubber and the silver tubes were in the ventricle, but the other end of the rubber tube had in some way become buried in the brain substance, though it was certainly placed in the subdural space. There were several thin adhesions of pia mater to dura mater over the vertex. The foramen of Munro was much enlarged, so that there was a very fine communication between the ventricles.

As there must be some doubt as to when exactly the hole, one third of an inch in diameter, formed between the ventricle and the subdural space, I will not refer further to it as a means of communication between the two. But the rubber tube put in on December 19th had certainly kept up such communication for more than two months, and yet during that time the hydrocephalus had steadily increased. There was nothing to account for the slightly greater increase of the left side, for with so large a foramen of Munro the ventricles might be regarded as practically one.

(*Read at the Provincial Meeting, Bristol, June the 18th*, 1904.)

Discussion.

Dr. James Taylor regarded Dr. Michell Clarke's case as extremely interesting. It was not often that one saw a case of recovered hydrocephalus, even after surgical measures. One point which should be dwelt upon in regard to those cases which recovered was that after recovery the child was often extremely

sharp, and even above the average in intelligence and in its power to learn quickly. He would be glad to know if anyone present could give any explanation of that fact.

Dr. G. A. Sutherland regarded cases of hydrocephalus as of extreme interest, because they were not at all uncommon, and, so far, the results of treatment had been unsatisfactory. From the clinical, and perhaps the therapeutical, point of view, he thought the cases should be divided into congenital and acquired. Speaking generally, one might say the congenital cases were due to syphilis acting during intra-uterine life. The acquired cases were very largely due to meningitis occurring in early life, and more especially that form known as basilar meningitis in infants. Dr. James Taylor had mentioned that it was rare to see hydrocephalus cured. If he meant at the age at which the present patient suffered he, Dr. Sutherland, thought perhaps he was right, but in early infancy he thought a considerable number were cured after a full course of mercury. With regard to acquired cases, he did not think medical measures had any influence upon them, save in very exceptional cases. Strapping, external drainage, aspiration had all been tried, but he thought that they had failed entirely in the vast majority of cases. In conjunction with Mr. Watson Cheyne, he had worked at the subject for a time, and, as a result of reading Dr. Leonard Hill's experimental work, they found there was a possible channel of drainage from the subdural space, as Hill pointed out that there was never any higher pressure in the subdural space than in the surrounding veins, and that any excess of fluid would be removed by absorption into the venous channels. They thought that there was a possible means of relieving hydrocephalus by means of subdural drainage. Their first case was a surprising success. The operation was over in a few minutes, and there was no subsequent disturbance. The skull went on diminishing in size, and for three months the child lived comfortably. The child eventually died, he thought from the syphilis from which it was suffering. There were some points which showed whether it was a practical method of treatment with regard to intra-ventricular pressure. The skull diminished so much in size that the frontal and parietal bones were largely overlapping; absorption of the fluid was so marked that the skull was reduced to its smallest possible capacity. After death it was found that very little brain tissue was left, that the brain had never properly developed, and that there was considerable meningitis about the base. Mr. Morton mentioned that after the operation the head seemed to increase in size again, although the drainage was free. Theoretically he thought physicians and surgeons were right about the possibility of curing hydrocephalus by subdural drainage, but he did not think that surgeons had yet attained to that success which probably awaited them in the future. The difficulties

seemed to be purely mechanical, and he hoped Mr. Morton would persevere in his efforts. If he could carry out really permanent drainage he would probably succeed in relieving the condition. At present, if the patient had a thick brain it was impossible to keep up the drainage, but it was possible with a bladder brain. In the latter case the operation did no good, because the brain was already so damaged that its development was rendered impossible. It was in the early stage of acquired hydrocephalus that they might hope for cure by operation, and he invited the co-operation of surgeons in overcoming the mechanical difficulties of drainage.

Mr. Clement Lucas said he understood from the excellent speech of Dr. Sutherland that he was chiefly responsible for this operation for hydrocephalus having been introduced. He (Mr. Lucas) had all along thought that surgeons ought to cure hydrocephalus, because it was, for the most part, a purely mechanical affection. In cases which he remembered the aqueduct of Sylvius not infrequently ended blindly, and was so dilated at its commencement that the little finger could be introduced into it. In other cases there was a blind ending at the base of the fourth ventricle, which did not communicate with the subarachnoid space. He had attempted operation in these cases years ago, and it had occurred to him that if the hydrocephalic fluid could be drained underneath the scalp, a communication being made between the ventricles and the under surface of the scalp, the fluid would be absorbed there. He accordingly cleared a space underneath the scalp, and made the space communicate with the ventricle by puncturing through the anterior fontanelle into the ventricle. So far it succeeded well, perhaps too well, for he got a tumour under the scalp as large as a turkey's egg, and he thought that with a little aid from pressure and massage it would be absorbed. But it remained there permanently and seemed to increase in size. After a time he felt compelled, therefore, to drain it. His next idea was to have a very oblique drainage, so that the fluid could be let out on the side of the head, and in that way there should be no direct communication with the ventricle. He made an incision some distance off, and drained the tumour through that opening. There came a difficulty in keeping the opening free, and he was compelled to put in a drainage-tube. The head became much smaller, but he could do nothing more, and the effect was very little different from ordinary direct drainage through the fontanelle. Eventually the patient in the bed next to the child had erysipelas, which the child caught, and died of meningitis. It had occurred to him that an attempt might be made to establish a communication between the ventricles and subarachnoid space at the base of the brain. He thought advances would be made in that direction,

for it was only necessary to let the fluid communicate sufficiently freely with the subarachnoid space to effect a cure.

Dr. George Carpenter felt sure that, as Dr. Sutherland had remarked, many of the cases were syphilitic, and he (Dr. Carpenter) had cured quite half a dozen of them by mercury. Dr. Sutherland had shown a very marked specimen of craniotabes, which he (Dr. Carpenter) looked upon as a syphilitic manifestation. He thought, after Mr. C. A. Morton's interesting case and its sequel, that free drainage into the subdural space was not a promising operation.

Mr. C. A. Morton (Bristol) (introduced), in reply, said that Dr. Sutherland had stated that if drainage could be maintained in these cases more encouraging results would be obtained. He (Mr. Morton) had brought the case forward because he thought it was clear that he had succeeded in maintaining the drainage for two months. Fluid flowed through the tube at the time of operation, and the end of it was not found buried in adhesions at the post-mortem, and the lumen of the tube was then patent. He would not say anything about the large hole which had formed between the ventricle and the subdural space, because he did not know when it was formed. He did not feel encouraged to undertake the operation again. He agreed that the metal tube used was obviously too small, but he did not think the rubber tube was too small for fluid had been seen to flow freely through it from the ventricle. With regard to the pyrexia it was very interesting that it came on so late in the case, *i. e.* the tenth day, instead of immediately after the operation.

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93. CASE OF DOUBLE CONGENITAL DISLOCATION OF THE HIP UNDERGOING TREATMENT BY THE LORENZ METHOD ("BLOODLESS").

By J. LACY FIRTH, M.S.

(Introduced.)

State immediately before operation. — Female, aged 4 years. There was excessive lordosis on standing and walking. The top of each trochanter was situated about three quarters of an inch above Nelaton's line. The heads of the femora were palpable on the ilia above and behind

the trochanters, and could be made to descend a little with traction.

Operation (January the 28th, *i. e.* nearly five months ago).—Attempts were made to reduce the dislocations by manipulation. The manipulations were, in the main, those described by Lorenz. At the conclusion it was thought that probably both dislocations had been successfully reduced. Plaster-of-Paris splinting was applied with the limbs in a position of extreme abduction and extension.

The splint has been changed twice, and that in which the child is shown to-day was applied a week ago with the limbs in almost the same position as in January, but with rather less abduction.

The measure of success with which the above described procedures have been carried out may be judged from an inspection of the skiagram shown, which was taken by Dr. Kenneth Wills. Another skiagram shows the condition of the hips before the operation.

The right hip seems to be satisfactorily reduced. The left femoral head appears to lie a little below the acetabulum.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th,* 1904.)

Discussion.

Mr. A. H. Tubby said he had many points to mention, but as the hour was late he would come to the point at once. He had been acquainted with the history of the so-called Lorenz treatment of congenitally dislocated hips from the commencement, and he possessed much of the literature of the subject, from the time when Lorenz commenced the open method of treatment up to the present date. He would not say much about the open method, but the so-called bloodless method was not entirely Lorenz's. It was a modification of a method which was brought forward by Paci, of Pisa, in 1894, the only difference being that Lorenz is more violent than Paci. Those who knew the literature of the subject would not assign the entire credit to Lorenz for the operation. Two months ago Dr. Ridlon, of Chicago, sent him (Mr. Tubby) a pamphlet detailing his experiences. Mr. Tubby had also made himself acquainted with the experience of other American surgeons. As Dr. Ridlon

so well said his first stage was one of curiosity, the second stage was one of delirium, and the third stage was the stage of reflection. Of course, no surgeon believed for a moment what the newspapers said on the subject, but there was no doubt the new method was not going to effect a great number of cures. It would only bring about cures in comparatively slight cases; in fact, in those cases in which the head was quite near to the acetabulum and a good shape, and the acetabulum was widely open. Therefore the method he thought was not applicable to all purely congenital cases. It was useful in those cases in which the child had developed a rather wide acetabulum, and where the head had slipped out at the moment of birth owing to the traction by forceps or by the legs, and then only if the case were seen before the fourth year. When the method was first introduced it was thought that a clicking sound indicated the entry of the bone into the socket. But since the forcible method had been employed surgeons had not heard the click with so much rapture. The click used to be regarded as diagnostic, and bystanders were asked if they heard the "click." As a matter of fact, it meant in many cases that the head had not gone in, but had slipped over a bony prominence or had changed its position from the back to the front of the acetabulum. The results, so far as they had gone, might be classified under three heads:—(1) Actual reposition; (2) good or fair results; (3) no result at all. He could not state the actual number of repositions, but it was comparatively small. The results which were said to be "good" or "fair" were those in which the posterior displacement had been replaced by anterior displacement, but without complete reposition. Such patients walked better, some of the shortening disappeared, and they lost their lordosis. Until the advent of the X rays many such cases were put down as complete cures, whereas they have since been shown to have altered from posterior to anterior dislocations. In other cases no good had been effected. Mr. Tubby had done the operation several times, and his own impression was that the operation should not be vaunted as a cure, and where anything like excessive force was called for it was the surgeon's duty to desist, because when force was called for it was almost certain to result in a bad dislocation or an untoward accident. No surgical procedure which might involve either fracture of the neck of the thigh bone or its shaft, or resulted in gangrene of the limb or rupture of the sciatic nerve, was worthy the name of surgery, and anyone who advocated that form of surgery was without his sympathy.

94. A CASE OF EXTROVERSION OF THE BLADDER.

By PAUL BUSH, C.M.G.

(Introduced.)

Mr. Paul Bush showed a child with extroversion of the bladder. A portion of the mucous membrane had "skinned over" under constant application of saturated solution of boracic acid. He proposed doing the old operation of turning over an upper central flap and covering this by sliding two lateral flaps over it. He pointed out that in this case there was no prepuce.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th*, 1904.)

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95. A CASE OF DOUBLE HARELIP AND CLEFT PALATE.

By PAUL BUSH, C.M.G.

(Introduced.)

Mr. Paul Bush showed an extreme case of double harelip, with protruding premaxillary bone and cleft palate.

He said he showed the case because he intended to operate in a few days, and he thought he would like information from his surgical friends as to whether it was a case for removal of the intermaxillary bone.

(*Exhibited at the Provincial Meeting, Bristol, June the 18th*, 1904.)

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96. VALVULAR OBSTRUCTION IN THE URETHRA OF AN INFANT, WITH SECONDARY CHANGES IN THE URINARY ORGANS.

By C. A. MORTON.

(Introduced.)

Although there are many specimens in museums of conditions somewhat resembling the one present in this case, clinical records of such cases seem to be rare. There is also one very important fact illustrated by this case, *i. e.* that the obstruction being valvular, with the valve sloping towards the bladder, a very serious obstacle may be present to the passage of urine, and yet a catheter may enter the bladder without the slightest difficulty.

A baby aged 13 months was sent to me at the Children's Hospital with a history of three months' wasting and vomiting, and a distended abdomen. This distension was mainly due to flatulence associated with hard scybalous masses, but a central hypogastric swelling suggesting a distended bladder was also present. The infant passed urine in apparently natural quantity, however, and I did not employ a catheter for a day or two after admission. When I did so I used a Jacques No. 3, and this entered the bladder quite easily, and withdrew several ounces of urine, and caused the hypogastric swelling to disappear. Blood and pus were noticed in the urine during the next twenty-four hours, and the temperature rose considerably. The temperature had been 101° F. and 102° F. after admission before the catheter was passed, and probably pus had also been present in the urine at that time, though I have no note of it. A large fæcal mass had to be cleared out of the rectum, and also scybala higher in the bowel were removed with enemata. The infant died a week after admission. The catheter passed was boiled, and every precaution taken to prevent infection by its means, and I

think there is no doubt that cystitis and pyelitis were present at the time it was used.

At the post-mortem a dilated and greatly hypertrophied bladder was found; the ureters were so dilated as to resemble small intestine; and both kidneys were much enlarged from hydronephrosis, but a considerable amount of renal tissue remained. The mucous membrane of the bladder was plum coloured from hæmorrhage, and had exudation of lymph on it: hæmorrhages were also present in the mucous membrane of the pelvis of each kidney, which contained purulent urine. A small valve-fold was found in the roof of the urethra just beyond the vesical orifice, directed downwards and backwards, so that although the passage of urine would be much interfered with, a catheter would not be obstructed.

(*Read at the Provincial Meeting, Bristol, June the* 18*th*, 1904.)

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97. THE WIGHTMAN LECTURE ON THE HEREDITARY BIAS AND EARLY ENVIRONMENT IN THEIR RELATION TO THE DISEASES AND DEFECTS OF CHILDREN.

DELIVERED ON JULY THE 15TH, 1904.

By R. CLEMENT LUCAS, B.S.

Mr. Chairman and Gentlemen,—In accepting the courteous invitation of the Council of the Society for the Study of Disease in Children to deliver the first Wightman lecture I feel that I have incurred a double responsibility, for it rests with the first lecturer to give a short history of the circumstances which have led the generous founder to establish this annual address and then to further his wishes still more by attempting to stimulate the observation and study of those diseases which are peculiar to children, or

of such as are modified in their effects by the presence of immature and developing structures.

I may state that my qualification for this office rests on the fact that, knowing all the circumstances, I can best interpret the founder's feelings, born of the anguish caused by the death of his only son, which have led him to endow this lecture: first, as a permanent memorial to his heir, in the sincere and fervent hope that other parents may be spared the trials he has gone through; and secondly, recognising how modern discoveries have lessened the terrors of the disease from which his son died, he trusts that future scientific observations may lower the mortality of other diseases to which children are liable and that this lecture may be the means of establishing or of diffusing such knowledge.

Alexander Cockburn Wightman, the only son of Mr. Charles Wightman, of Portland Place, was born on August the 14th, 1877, and died from diphtheria on August the 16th, 1884, having thus just completed his seventh year. The life history of a child dying at this early age lies engraved for the most part in the hearts of his loving parents, and rests concealed within their breasts. To attempt to wrench it thence and expose it to public gaze involves a cruelty from which I have shrunk, yet I feel that in leaving much unsaid that might have been gained from these sources I may, perhaps, do scant justice to those good qualities of heart and mind which this little fellow so early manifested. Bright, affectionate, and unselfish, he was a centre of intense happiness within his family circle; intelligent beyond his years, and perhaps beyond his strength, he gave promise of future high mental development; whilst his warm and impressionable nature was deeply "moved with concord of sweet sounds," and music became his favourite pastime. Many little considerate acts proved his sweet unselfish nature, and endeared him to all who knew him. He became the sunshine of his home. But a dark cloud overshadowed it when, in August, 1884, he was seized with a malignant

form of diphtheria, associated with a croupy cough and intense albuminuria. Tracheotomy became necessary to relieve his breathing on the second day, and afforded him immediate relief; but twenty-four hours later cyanosis again occurred, and was again relieved by removing a quantity of false membrane from the trachea; after this uræmic convulsions ensued, and in one of these attacks, about forty-eight hours after the operation, he ceased to breathe. Such is the brief history of this short life so abruptly closed by a disease the bacillus of which at that time was unknown, and the serum antidote for which was not discovered till some ten years later.*

It has been said of great reformers and of leaders in science, such as Copernicus and Galileo, that they lived before their time, because their views and discoveries were not sufficiently appreciated during their lives; but far more truly may it be said that many children, such as little Wightman, have died before their time, simply because the results of discoveries subsequently made were not then available to check the progress of the diseases to which they succumbed. Those who are said to have lived before their time have their fame revealed and their monuments engraved by succeeding generations, but what is left to those who die before their time? What might have been for them to accomplish is lost for ever in conjecture. The developing frame, the expanding mind, the deepening affections, all are lost, and there remains but a

* The bacillus of diphtheria was first described by Klebs in 1883 as an organism to be found in the diphtheritic membrane, but it was not till a year or two later that Löffler cultivated it in artificial media and proved its communicability by inoculation, since which time it has been called the Klebs-Löffler bacillus. Behring began his experiments with serum in 1890, but it was not till 1894 that a satisfactory serum suitable for the human subject came into use. Herringham draws the following contrast between the cases before and after the introduction of the serum treatment at St. Bartholomew's Hospital. During 1893 and the first part of 1894 95 patients were treated under ten years of age with 50 deaths, or 52·7 per cent. From July 1st, 1894, to July 3rd, 1896, 50 patients were treated with antitoxin serum, of whom eight only died or 16 per cent.

vision of the past. No one can interpret what can never come to pass, and the hidden forces of potential energy cut short by death are lost for ever. But there is left the record of a short life of unsullied innocence, such as led the great satirist of nearly 2000 years ago to proclaim the apparent paradox that the greatest reverence is due to a boy—*Maxima debetur puero reverentia.* With these few words I pass to the second part of my task, and as we are in the initial stage, both as regards our Society and its relation to this lecture, it may be well to start with the beginning of things, and I have selected as my subject "The Hereditary Bias and Early Environment in their relation to the Diseases and Defects of Children."

I will say at once that it is not my intention to discuss the various theories of heredity, the "pangenesis" of Darwin, the "physiological units" of Herbert Spencer, or the "ancestral germ-plasm" of Weismann, not to speak of the various modifications of such theories which have more recently been suggested the better to accord with recognised facts; but we shall treat the subject from a purely clinical point of view. We start with the general axiom handed down from all time that a child must in many general characteristics resemble his parents; his height, his complexion, his features, his hair will so far be determined by his parentage that strangers are usually struck by a certain family likeness. When there is a wide distinction in appearance between the parents it is often easy to trace the preponderating influence of one or the other of them, and this is often suggestive of the relative vigour of the father or the mother. Thus a peer who married a lady many years his junior had a family of eight children, only one of whom carried the height, features, and complexion of the father, all the others resembling the mother. It is not improbable that the relative vigour of the parents may account for the variation of individuals of a family as well as for that reflected resemblance which may often be traced to the ancestors of one or other parent.

At first I would speak of a peculiarity, not infrequently met with, where the strength of the family is found centred in one or other sex. This is to some extent illustrated by little Wightman, for he had a sister exposed to precisely the same influences, who suffered at the same time, but who recovered without presenting any serious symptoms; and relative delicacy of the males compared with the females had been noticed in other branches of his mother's family. Instances might be quoted without number of families in which there is a great preponderance of one sex, whilst the individuals of the other sex are delicate and die young. The centenary of Cobden reminds me that Richard Cobden had an only son, who died at the age of fifteen years, and five daughters, four of whom are living at ages approaching or past sixty years. When the strength of the family is concentrated in the female line we meet with a common cause of the extinction of titles. I take haphazard the Barony of Rivers. The last peer died in 1880 at the age of nineteen years, but his four sisters are all living at advanced ages. Some regard the excessive production of girls as an indication of a weakening of the reproductive function, and it has been asserted that a common cause of the extinction of titles is due to the holders marrying heiresses for the sake of their fortunes.* Instances might readily be quoted of families in which there was a large preponderance of boys and in which the girls were delicate or died early. It may, I think, be regarded as generally true that when there exists a very strongly marked tendency to the production of one sex the other sex presents less vitality.

Another general characteristic which is distinctly hereditary is longevity, which may be translated into a power of resistance to disease; and as its antithesis, brevity of

* Galton, taking the legal peerages, showed that marriage of an heiress caused extinction of the titles in 16 out of 31 peerages. Again, he found 14 instances in which the eldest son of a first peer married an heiress, resulting in eight cases of absolute sterility and two of only one son.

life, tends to obliterate itself, length of life with the aid of selection and hygienic surroundings ought gradually to be extended, and this recent statistics have proved to be taking place.

When we inquire what diseases may be regarded as hereditary we meet with a difficulty at the very threshold of our inquiry, for diseases formerly thought to be constitutional and hereditary are yearly diminishing under the light of modern discoveries. It is interesting to look back and note what diseases were thought to be hereditary forty or fifty years ago. Thus Dr. W. Sedgewick, writing on this subject in 1863, gives pityriasis versicolor of the chest (long known to be a fungus disease) attacking the males of a family—father, sons, uncles, and cousins—as a hereditary disease. A hereditary horror of soap and water might possibly have existed in this family and led to the spread of this easily destroyed fungus among the various members. Those who were brought up to a careful study of diathesis as a cause of hereditary disease will remember what a shock was given to all preconceived ideas of tubercle by the discovery of Koch's bacillus. It is interesting to read how this deeply impressed idea of a diathesis or constitutional disease caused physicians before that time to shut their eyes to the most obvious facts. Let us turn to that most eloquent writer, Sir Thomas Watson, whose book on medicine so long held sway as the classic authority, and read what he says on phthisis. He asks, "Is phthisis contagious? No. I verily believe it is not. A diathesis is not communicable from person to person. Neither can the disease be easily, if at all, generated in a sound constitution. Nor is it ever imparted to another, even by one scrofulous individual to another." Then he goes on to explain away the obvious cases of contagion: "A wife watches the death-bed of her consumptive husband, and presently sinks herself under consumption, and there may be no tubercle or acknowledged examples of scrofula in her pedigree. Yet even here the latent diathesis may fairly be presumed to have

existed. Very few families are perfectly pure from strumous intermixture. The predisposition may be slight, it may be dormant for a generation," etc. This shows how a predominant theory may close the eyes of distinguished observers to obvious facts. When I became attached as Surgeon to Out-patients at the Evelina Hospital for Children I made careful inquiry as to the family history of every case of early tuberculosis that came under my observation. These were cases of tuberculous abscesses of skin and cellular tissue, tuberculous glands, dactylitis, bone and joint disease, tuberculous epididymitis, and lupus. My cases went to show that in cases of infantile tuberculosis occurring under the age of two years one or other parent was nearly always found suffering from phthisis, and in some few cases where both parents were suffering from phthisis the disease began early and generally proved rapidly fatal. These observations were made to prove the effect of diathesis and double diathesis on the offspring, but the obvious cause now easily understood was the atmosphere charged with tubercle bacillus in which the infants resided. Are we then to throw over tuberculosis altogether as a hereditary disease? We know that any type of person sufficiently depressed in health and inoculated with or exposed to Koch's bacillus may acquire tubercle—the short, the tall, the fair, the dark, the ugly, and the good-looking. But were the old observations as to the particular type prone to phthisis all wrong? Those who think so declare that the type described was formed of persons already suffering from tubercle in whom the refined features, slim figure, and pearly sclerotics only indicated a wasting disease that had commenced its work.

Others regard the type or diathesis as indicating merely a hereditary weakness of resistance to the invasion of the tubercle bacillus. But this opens up another question, may not the type have been produced by the effect of the disease in preceding generations? Phthisis is a chronic disease which may go on for years. By consolidating the

lungs it causes contraction of the chest. By diminishing the cellular tissue it gives refinement to the features and causes the eyes to appear larger. By diarrhœa and perspirations it clears the complexion. By obstruction to the circulation it gives a peculiar shape to the finger-nails. Now may not the variation in appearance thus brought about be impressed on succeeding generations? If so, the type of the diathesis becomes the *hereditary effect* not the *hereditary cause* of the disease.*

There is yet another view of the transmission of tubercle suggested by Cohnheim, but developed by Baumgarten, that the bacillus actually penetrates the germ or sperm cell and is in this way directly conveyed to the next generation, being held quiescent by the rapid development of the cells of the ovum, but subsequently during the life of the growing or fully-developed animal multiplying and producing its effects. It is founded on the analogy of the pébrine disease of the silkworm, the ova of which Pasteur showed became infected with the parasite yet hatch normally, but the caterpillars produced die subsequently as the result of the growth of the parasite within them. This view is supported also by the experimental introduction of the bacillus into fertilised hen's eggs, in which way tuberculosis in chickens has been produced. Until, however, the bacillus is actually seen within the germ or sperm cell this suggestion must remain a somewhat improbable theory. If true it leaves the future of the child born of tuberculous parents hopelessly handicapped from the earliest phase of his existence. On the other hand, inoculation through some abraded surface, or infection through the air, or the tuberculous milk of the mother or a bovine substitute, are conditions all-sufficient to account for early tuberculosis. If the disease be produced in this way a far more hopeful horizon is opened up for the child of tuberculous parents. Change of environment, removal from the rooms infected by the parents to fresh mountain

* This is a Lamarckian argument which would not be admitted by the followers of Weismann.

or sea air, and pure nutritious diet give a reasonable hope that the child may escape the disease and grow up a useful member of society.

Another disease, which through ages has been regarded as hereditary is leprosy, a disease so ancient that it was described in the "Ebers papyrus" 1300 years B.C., and prescriptions for the cure of it are said to date 2000 years B.C. Its contagiousness was also recognised or believed to exist from the earliest times, so that lepers became outcasts. Segregation has been for centuries the means adopted to prevent the spread of the disease, and is still enforced with greater or less stringency in leprous countries. That leprosy was believed to be hereditary in the days of the Hebrew prophets may be gathered from Elisha's curse on his servant Gehazi, "the leprosy therefore of Naaman shall cleave unto thee, and unto thy seed for ever." Up to quite recently its heredity was a prevailing belief among medical men; but the Indian Commission failed to find histories of the disease in the families of more than 5 or 6 per cent. of the lepers examined, and, what is still more remarkable, Hansen, who went to America to trace the descendants of 160 Norwegian lepers who had emigrated to that country, found that not one of their descendants had inherited the disease. Since the discovery of the *Bacillus lepræ* by Hansen in 1871, the cause of the disease has been made clear and its inoculability rendered highly probable. True, experimental inoculations have rarely been successful and the lower animals appear to be immune, but that the bacillus found in the characteristic nodules is the cause of the disease there can be no reasonable doubt. The manner in which the disease is spread remains to be elucidated, but this is only a matter for further observation. How few years have elapsed since the organism of malaria was shown to be carried by the anopheles mosquito. Yet no educated man can now doubt the evidence resulting from the exact scientific observations which demonstrated the origin and mode of distribution of that disease. When it is once

clearly shown how the *B. lepræ* is introduced into the body, all discussion as to hereditary transmission of the disease will cease. It may be, as indeed was suggested by Virchow, that certain dispositions may show less resistance to this bacillus as certain persons show less resistance than others to the bacillus of tubercle, and that certain families in this way may be more prone to the disease than others. But the children born of leprous parents are not leprous, and if early separated from their parents rarely become so. To save the children it would seem as urgent, as in the case of children born of tuberculous parents or even more so, to remove them at once from their unhealthy surroundings to a district free from leprosy. I cannot end my remarks on this disease without paying a tribute to the highly scientific investigations of this scourge by my old friend and pupil, the late Dr. Beavan Neave Rake, who fell a martyr to this horrible malady as the result of devoting his life to the alleviation of the sufferings caused by it among the natives of Trinidad.

After what I have said of leprosy I need scarcely more than allude to malaria, which in lectures delivered before the Royal College of Surgeons in 1884 was described as a climatic diathesis. The protozoon was discovered by Laveran in 1880, and its life-history has since then been worked out—its inoculation after sunset by the night-feeding anopheles, and its destruction by quinine. Its heredity, therefore, need not be discussed, but the effects of malaria in infancy and childhood are most injurious, not only on growth and development, but especially on the mental faculties of the child. Protection by avoidance of exposure after sunset and by quinine and early removal to a non-malarious district should be insisted on for the healthy rearing of children born in malarious districts. With tubercle, leprosy, and malaria taken out of the diathesis and hereditary disorders by the discovery of their respective parasites and classed under infective diseases

the belief in general hereditary diseases has been very much shaken.

We pass now to the consideration of a disease which we know can be transmitted by either parent to the offspring during the active stage of the disease. I allude to syphilis. From one generation to the next it passes, but beyond that transmission is very doubtful. I doubt whether a disease propagated only so far deserves the name *hereditary*, and I am inclined to suggest that the term *transmitted syphilis* far more correctly expresses our knowledge of this disease in childhood than the term *hereditary syphilis*, which is commonly employed. This disease, so aptly described by the late Dr. W. Moxon as a "fever diluted with time," becomes transmissible owing to its lengthened period of activity, but the exact process of transmission will remain obscure and to some extent theoretical until the actual organism causing the fever, be it protozoon or bacillus, is discovered. At present we only know it by its effects. Mr. J. Hutchinson, whose clinical researches in this disease cannot be too highly praised, regards the disease as capable of being carried by either the sperm or germ-cell to the fœtus, or by the mother to a healthy fœtus if she be infected during her pregnancy. What is certain is that if both parents suffer severely from the disease the results to the offspring are doubly disastrous. I have notes of a case of this kind in which the first eight conceptions ended either in miscarriage, premature births, or the births of children that only survived a short period, and it was not till the ninth that a child was born with sufficient vitality to live through the first two years.

In criticising the term *hereditary* in its relation to infantile syphilis I mentioned that it was doubtful if the disease could be transmitted to a third generation. I have notes of only one case bearing on this subject, but it is a very strking one. A blind man met a blind girl at a school for the blind and they married. They were both characteristic specimens of transmitted syphilis,

having notched teeth, opaque cornea, square heads, and scarred mouths. The man was admitted under my care for syphilitic bone disease, and, hearing that his wife had an infant two months old, I sent for it. The child was not a fine one, but she was quite healthy, and during the time I watched her, more than a year, she showed no signs of transmitted disease. Mr. Hutchinson says that about eight cases have come under his observation in which persons undoubtedly the subjects of inherited disease have become parents. With one doubtful exception, he had never been able to discover any evidence of disease in the offspring.

Syphilis is a disease over which drugs exert a powerful controlling influence, and under favourable surroundings and good diet it tends to wear itself out. In comparing syphilitic with tuberculous families it will generally be found that the later children of syphilitic parents are the most robust, whilst the elder children of phthisics, that is those farthest removed from the later stages of the disease in the parents, are the most healthy. Syphilis, although apparently not transmitted as an active disease to the third generation, nevertheless has such an effect on the developing infants of the second generation as to lower their general physical type, which is shown in stunted growth, flattened bridge of the nose, imperfect teeth, coarse skin, and general defective development. The new type thus formed will have a tendency to be transmitted, and by intermarriage to be rendered more permanent, and this disease thus becomes one of the causes of physical degeneration in large cities.

Another disease more especially occurring in large towns, and responsible, but to a less extent than syphilis, for physical degeneration is rickets. So far as our present knowledge carries us this is a purely dietetic disease, but it can be transmitted both as a tendency and as a type. It can be produced in any child by withholding proper nourishment and substituting an innutritious diet. Ignorant artificial feeding lays the foundations of rickets, which

first shows itself in the over-distended abdomen, perspirations, capricious appetite, and irregular bowels; then follow the enlarged epiphyses, the bending of bones, the softened ligaments, large head, and widening between the eyes, beaded ribs, and L-shaped thoracic depressions. It is a common disease in underfed puppies, and by breeding from rachitic types the dachshund has been specialised with its large head, long body, short bent leg and thigh bones, and splay feet. The most approved type of field spaniel seen at dog shows is also a good specimen of hereditary rickets. The general effect of rickets is to enlarge the head and shorten the stature. So long ago as 1881, in a discussion at the Pathological Society, I suggested that it might be justifiable to induce rickets artificially in microcephalic idiots and small-headed infants with a view of giving more room for the brain to develop. I think it may be said that persons who have recovered from rickets and have afterwards been well nourished often show great capacity for learning, so that this disease offers some compensation in greater skull capacity for the inconvenience of bow legs or knock-knees, all of which may be corrected by surgical treatment. But a child that has been the subject of rickets never attains to what should have been his proper height.

The two diseases, syphilis and rickets, the one dependent on uncontrolled vice and the other on injudicious or insufficient feeding and bad air, are the chief causes of the stunted growth and weak physical development of the lower orders in large towns. We have only to watch an organised demonstration of those out of work march through the streets of London to have brought home to us the wretched specimens of humanity reared in the slums and alleys of this great metropolis. Their poverty is seen to be obviously due to incapacity, their incapacity to defective development, and their defective development to moral and physical degradation. Every encouragement should therefore be given to those societies whose work it is to rescue children from the pernicious and unhealthy

surroundings of towns and bring them up in the fresh air of the country.

Rheumatism is another disease, or group of diseases, which has been ranked as a diathesis and regarded as hereditary. Some believed rheumatic fever, rheumatoid arthritis, and gout to be simply phases of the same diathesis at different periods of life. Others with clearer insight recognised two distinct diatheses, a rheumatic and a gouty. But rheumatic fever runs a course with definite symptoms, like an acute specific fever, and now that a diplococcus has been found in the rheumatic nodules, joints, and vegetation on the valves, which can be cultivated and be used to reproduce the disease in the lower animals, this disease will soon be classed with other specific fevers, and the idea of its being hereditary will disappear. There seems, indeed, to be evidence of "rheumatism houses" as there are "tubercle houses" and "cancer houses," probably low-lying, badly-drained places, where such organisms as cause the diseases, once introduced, continue to flourish. I am inclined to think that the history of joint diseases will soon have to be rewritten entirely, and the so-called rheumatisms will be classified as inflammations of joints dependent upon this or that organism. We already know that the gonococcus, whether introduced through the conjunctivæ or urethra, may cause a specific inflammation of a joint in which the coccus can be traced. We know that the pneumococcus, bacillus coli, streptococcus, and staphylococcus may all attack the synovial membranes, and probably there are others, as yet undistinguished, which induce special inflammations.

When we come to gout we recognise another dietetic disease similar to, or rather the antithesis of, rickets. Gout like rickets can be produced in a single generation, and again like rickets it shows a tendency to be reproduced in succeeding generations. Rickets is caused by the under-feeding of infants, gout by the over-feeding of adults with the addition of fermented liquors. In considering the heredity of gout, which is said to exist in

three fourths of the families of the leisured class, it is necessary to remember that the children are brought up, as a rule, in the same affluent circumstances as their parents, and are early taught to live in the same way. The type of man usually regarded as gouty is illustrated by the John Bull of 'Punch'—a plethoric, heavy, genial, but somewhat irascible individual, with a very good opinion of himself; but the type does not always breed true, for there are many thin, wizened, little old men who are martyrs to gout.

In taking a general view of the so-called diathesis and their formerly believed heredity one finds that modern discoveries have greatly disturbed the views formerly held as to the influences heredity was supposed to exert in the causation of disease. Tubercle, leprosy, malaria, and I think I may say rheumatic fever, are all infective diseases, the micro-organisms of which are recognised. Syphilis when active may be transmitted to the second generation, but there its influence as such probably ceases. Rickets and gout, the two dietetic diseases, appear to exert a stronger influence and to carry their effects further. Gout with its sodium biurate deposit is the sole survivor of the chemical theory of disease. But there may be yet further surprises in store for us. It may be that this is not a mere crystallising out of a super-saturated solution of the salt from the synovia, for if so all joints ought to suffer alike. Perhaps it may be shown that a particular micro-organism is favoured by the presence of a highly nitrogenous and alcoholic medium and is the cause of the precipitation; and that in rickets the mal-assimilation may favour the growth of another organism, the presence or the toxin of which may cause the changes in the bones and epiphyses characteristic of that disease.

I ought perhaps to make a few remarks on cancer which, in spite of the strong evidence of its local origin, many have thought to be a hereditary disease. The late Sir James Paget strongly believed in its hereditary

character, yet was only able to discover a trace of the family history in one in three of his private patients and in one in five or six among hospital patients. John Birkett always maintained that it was much more common to find cancer commencing in a person who had no family history of cancer than in one who had, and Cripps showed that the proportion of deaths from cancer among parents and grandparents of cancerous patients was scarcely larger than the proportion of deaths from cancer to deaths from all other causes. Evidence seems to be accumulating that we are dealing with a disease that in certain circumstances is contagious and inoculable in the same species. Cancer in mice can be readily inoculated in other mice. It seems certain, too, that cancer attacks persons living in the same circumstances and in certain houses. I think I can claim to be the first to publish the particulars of three persons unrelated having cancer at the same time, in the same house, in a note to the 'Lancet' on April 2nd, 1904 (p. 929). A gentleman living in London had rodent ulcer of the face on which I operated several times. His wife had cancer of the breast from which she died some two years after operation, and the gentleman's partner, who lived in the same house, died from cancer of the tongue. I think there is much more evidence of contagion among persons of advanced age and weakened resistance subjected to local irritation than there is evidence of hereditary transmission.

When we pass in review defects of the nervous system we are compelled to admit that a hereditary disposition in these cases is often very strongly marked. In the case of idiots Shuttleworth and Fletcher Beach found in 21·38 per cent. hereditary mental weakness (insanity or imbecility) and in 20 per cent. epilepsy and allied neuroses, and parental intemperance in 16·38 per cent. Whether disuse of the brain in the parents may be regarded as predisposing to idiocy some probably would be inclined to question, but the late Langdon Down gives some interesting statistics concerning the children of the three

learned professions, law, medicine, and the church. Three per cent. were the children of lawyers, 4 per cent. the children of medical men, and as many as 18 per cent. the children of clergymen in his asylum for the children of the better classes. He explains this by supposing that for success in law there is required the greatest amount of mental and physical vigour, that in medicine the race is less urgent and the claims not so imperious, whilst in the clerical profession a moderate success is still more insured and there is less claim among the rank and file for great mental and physical exertion. Galton approached this question from the opposite standpoint. He took the members of the councils of the most prominent scientific societies of London and found that the legal profession furnished 11 per cent. of the foremost men of eminence. The medical profession gave origin to 9 per cent. of scientific men, whilst the clergy gave birth to only 6 per cent. of men of science. The gradation, being similar though not the same, renders these two observers' facts taken together extremely suggestive. To turn for a moment to a more inviting subject, that of genius, I think it is generally admitted that Galton in his book on 'Hereditary Genius' proved that where a genius had once been developed exceptional ability was apt to be shown by many of his immediate descendants, and in some instances for several generations. Havelock Ellis has attempted to show that there is a relation between genius and stature. Taking the average height of these islands as given by the Anthropometric Committee of the British Association as from 5 feet 4 inches to 5 feet 9 inches, he proceeded to make a table of all the recognised geniuses and found that 142 were above 5 feet 9 inches, or tall, and 125 below 5 feet 4 inches, or short, and only 74 were between these heights, so that he concluded that genius abhors the middle height.

There are some nervous affections which are very hereditary and are prone to attack individuals of a certain stock at a definite period in their lives, as if certain parts

of the nervous system owing to some original defect were liable to wear out before the rest. Mr. D. S. Gerrish at the Bristol Meeting of this Society held in June last, related the history of a family in which he had traced through five generations a special paresis always affecting the distribution of the anterior tibial and peroneal nerves, commencing between the ages of twenty-two and twenty-six, and leading to muscular atrophy. It attacked females as well as males, and he had traced it in sixteen individuals belonging to this family.

Galton and Karl Pearson have attempted to show that hereditary peculiarities are transmitted in definite proportions so as to be capable of being expressed with mathematical precision. Galton took stature as his standard to work upon, premising that what would be true of one quality, physical or mental, would be true of all. He came to the conclusion that hereditary peculiarities were transmitted in the proportion of one-third to a son, two-thirds to a daughter, two-ninths to a nephew, and one-tenth to a grandson. Pearson working at the resemblance of brothers found that the measure of these indicated by the well-known regression line, was as two to one,—that is to say, that if one of the pair exceeded the mean by a certain amount the other of the pair tended to exceed the mean by half the amount; and similarly in respect of defect from the mean. This was always true for all the physical characters yet worked out in man. He then extended his observations to ability and intelligence, and found as the result of many observations made on 1918 pairs of brothers that the index worked out almost precisely the same—*i. e.* as two to one. He concluded that the psychical characters were inherited precisely as the physical, and that though education might aid, it could never create, intelligence—"it must be bred."

Deaf-mutism is a condition which has been supposed to be attributable largely to in-breeding, but the evidence

as to this does not appear to be absolutely conclusive.* Certain it is that if a deaf-mute has appeared in a family the defect will almost certainly appear in other members of the family, among cousins or indirect descendants. Deaf-mutism probably depends on various defects, both structural and nervous, which may vary in transmissibility. Edward Cock found absence of semicircular canals, or cochlea, and closure of the fenestra rotunda, reversions to a similar type, whereas others have attributed the defect entirely to nervous lesions. It is often associated with idiocy in Switzerland, where in some cantons 1 in 206 is a deaf-mute.

One of the most remarkable of hereditary diseases is hæmophilia, which selects only the males on whom to produce its effects, yet it is only transmitted by the females of a family. Nothing apparently seems to be known of the pathology of this peculiar disease, but the mode of transmission is not peculiar to bleeders; for colour-blindness almost invariably follows the same law. Earle traced colour-blindness through five generations, and out of eight families there was only one in which the defect was not strictly limited to the male sex, but it was always transmitted by the females; nine-sixteenths of the males were affected, but only one-fifteenth of the females.

Polydipsia has been stated by Gee to follow the same law, attacking the males and being transmitted by the females. Of the various congenital defects of the eyes which have been shown to be hereditary, total absence of the iris, coloboma, and irregular pigmentation of the membrane have been noted in successive generations. Amaurosis and congenital cataract, external and internal strabismus, dependent on errors of refraction, are all liable to be transmitted. Nyctalopia, or day-blindness, and hemeralopia, or night-blindness, are both hereditary. Albinoes all more or less suffer from nyctalopia, and

* In-breeding, according to Shuttleworth and Langdon Down, simply causes a concentration of any particular tendency.

hemeralopia has been traced by Cunier through six generations.

If we now proceed to consider external defects and deformities we shall find abundant evidence of hereditary transmission through many generations. Of skin diseases ichthyosis is one of the most remarkable, and in the family of the celebrated porcupine man Lambert, who was exhibited at the Royal Society in 1731, the disease affected the males for four generations, and two of his descendants were exhibited in 1802. Psoriasis also shows a tendency to become hereditary, and Hutchinson has suggested that it may in this way become the parent of ichthyosis. The same peculiarity in transmission, as noted in hæmophilia and colour-blindness, has also in several cases been noted in regard to ichthyosis—namely, that it may attack only the males and be transmitted by the females.

The most remarkable instances of hereditary transmission through many generations are furnished by such deformities as supernumerary fingers and toes and suppression or webbing of digits. In the 'Guy's Hospital Reports' for 1881 I traced through five generations the development of supernumerary fingers and toes. A great-great-grandmother had eighty descendants, of whom twenty-four individuals, or 30 per cent., were thus affected. Two other deformities were also found in this family—viz. webbing of certain fingers and toes, and harelip and cleft palate. It would seem that in some cases these supernumerary digits have been reproduced after amputation, and even re-developed after a second amputation. I have never met with this experience in my own practice, but Darwin quoted three instances in which it occurred, and explained the recurrence as due to "reversion to an enormously remote, lowly organised, and multidigitate progenitor." It is known that the fins of fishes may be reproduced after amputation, and the salamander's feet and tail will grow again after removal even to six times. Not less tenacious of reproduction in successive generations is the suppression of certain phalanges resulting in short-

ening of the fingers or their complete absence. A case is quoted by Sedgewick in which this was traced through the female line for ten generations. Ogle described, in 1872, a case in which the first and second fingers and the second and third toes had only two phalanges, whilst the third and fourth fingers and the fourth and fifth toes had each only one phalanx. He traced this defect through three generations and through collaterals living in distant places, at Manchester and Lincoln. Tubby has published particulars of a family in which the feet were like lobster claws, the three central toes being suppressed, whilst the hands had each only one finger, the fifth. He traced this deformity through four generations, and, in spite of the admixture of new blood, fifteen out of twenty-two descendants were deformed. Many other defects and deformities which are hereditary I have only time to mention. Congenital dislocation of the hip has been transmitted through four generations. Absence of the patella has been noted in three successive generations. Webbing of the toes and fingers has been traced through three or four generations. Club-foot is often hereditary; so are harelip and cleft palate. In respect of this deformity it is interesting to note how a slight defect in one generation may foreshadow a more serious abnormality in a succeeding generation.

In the 'Transactions' of the Clinical Society for 1888 will be found a paper in which I showed that a congenital absence of the lateral incisor tooth was an indication that a harelip or cleft palate in the same plane might follow in a successive generation. I now for the first time make public a further observation in relation to this deformity. It is that a feebly developed lateral incisor on one side in the mother may foreshadow a harelip and cleft palate in her offspring. I have twice noted this association, and I exhibit to The Society a photograph and drawing of the teeth of a mother, showing this defective lateral incisor, whose first and only child has harelip. Harelip and cleft palate are frequently associated with other deformities which apparently present no causative relation. I men-

tioned that in the family with supernumerary fingers and toes which I recorded harelip and cleft palate had also appeared in the later generations. Roux has recorded a similar association. Cooper Forster recorded particulars of a family in which imperforate anus was associated in several instances with harelip and cleft palate.

Our 'Reports' already contain several instances of multiple congenital deformities. In the first volume Carpenter relates the histories of two sisters who presented many abnormalities and were of weak mental capacity. Each had a keel-shaped skull, congenital heart disease, abdominal ruptures, webbed fingers, and six toes on each foot. Jackson Clarke describes a case of absence of the radius causing club-hand associated with macrostoma. In the second volume Hutchison and Sutherland each relate a case of multiple deformities of almost identical characters. Two boys, one aged thirteen months and the other two years, presented the following abnormalities in common:—(1) Congenital heart disease; (2) rudimentary harelip; (3) six digits on each hand; (4) shortening of all the long bones of the extremities; and (5) defective development of the gums. The shortening of the limbs and the spreading of the fingers and toes resembled cases described as achondroplasia, but other defects were superadded, and Shuttleworth thought that their intellects were defective. In the third volume Lewis Marshall gives particulars of a family at Nottingham in which truncated fingers and toes had occurred for five generations, and in some of those affected double or supernumerary thumbs had been noticed. This family defect so closely resembles that described by Ogle in 1872 that one is almost inclined to think that some of the same family which Ogle found in London, Manchester, and Lincoln had found their way to Nottingham. I have not time more than to mention the tendency of partial hypospadias to be reproduced or exaggerated in succeeding generations. Hernia, varicocele, and varicose veins are often found hereditary, so is atrophy of one testis.

I wish now to make a few remarks on constrictions and so-called intra-uterine amputations, as they probably are closely related in cause and effect. The remarkable case shown at the Bristol meeting of this Society by Mr. H. E. Harris, in which there were complete absence of both lower limbs and of the left upper extremity, and webbed fingers on the hand of the only limb remaining, will sufficiently illustrate intra-uterine amputations; and the no less remarkable case of deep encircling constriction of the abdomen associated with a depressed furrow above the left knee published by Dr. C. O. Hawthorne in the third volume of our reports and in 'The Lancet,'* and another shown by Mr. H. J. Curtis of multiple dimpling and grooving of the surface associated with club-feet will illustrate the simpler condition. The most generally received explanation of those constrictions and amputations is that they are caused by the umbilical cord. I am inclined to think that there is little more to be said in favour of the constricting funis theory of intra-uterine amputation than there is for maternal impressions, which I will not waste the time of the Society by arguing against. If the funis were so tightly wound round a limb as to cause amputation, surely the counter pressure on the cord itself would stop the whole fœtal circulation and kill the fœtus. Again, if the funis ever causes amputation, considering the extreme frequency with which it is found round the neck of the newly-born child, amputation of the head would be one of the most common of the intra-uterine amputations. Then taking Harris's case, is it likely that a cord could first amputate both lower extremities at the hip-joint, and then proceed to amputate one upper extremity at the shoulder-joint? The number and the level of these three amputations completely dispose of the theory. If the funis amputated one it would thereby be so relaxed as to lose its constricting influence on the others. Even supposing it possible, the webbed fingers on the only limb still remain to be explained. Then what

* The 'Lancet,' August 8th, 1903, p. 374.

of the amputated fingers and toes? Will the funis in any way account for the loss of eight fingers and eight toes? Or, could it pick out the three central toes and take off the thumb and three first fingers of each hand, as in Tubby's case? Hawthorne's constricting abdominal cicatrix at first sight looks very like something that might be caused by the cord, but on closer examination it is seen that the constriction is below the level of the umbilicus and does not run up to it, as one would expect if caused by the funis. The grooving of the thigh and the double grooving over the loins to me suggest the probability of a general condition rather than the mechanical effect of the cord. Curtis's case is accounted for on the additional theory of amniotic adhesions, of which there is no evidence. The multiple dimplings and grooves are probably to be accounted for by errors in development similar to those which caused the clubbed feet present in that case.

I do not wish to argue longer in the negative. What theories can be put forward to explain these deformities which commence in a family and then cling tenaciously to subsequent offspring, in spite of fresh blood being added at every generation? They seem to suggest a defect very far back in the procreative process, and the frequency of many associated defects implies something very general. An incompatibility of germ- and sperm-cell, such as produces hybrid deformities in plants, appears to me a not unreasonable hypothesis as the cause of defects dating back to the earliest stage in development.

Next to this, considering the vast number of deformities which are associated with defects in the nervous system, as, in many of the cases I have mentioned in illustration, a defective nerve control will be a sufficient explanation of many defects occurring at a later period. Morphœa, or scleroderma, is often symmetrical, and by many is thought to be governed by nervous distribution. Such a condition, to my mind, offers a better explanation of intra-uterine amputations than any mechanical one of the cord. There is yet another way in which suppression of an extremity

might be accounted for—that is, by supposing a cutting off of the blood-supply through an endarteritis similar to the endocarditis which often closes the pulmonary orifice and is the cause of congenital heart disease, or plugging of the main artery by embolus, but this would scarcely alone account for the associated deformities such as webbed fingers and the like.

The most difficult of all deformities to account for are those in which there is an excess of the normal development, as with supernumerary digits, if Darwin's reversion theory be not accepted, but the frequent association of these with lack of completion in other parts indicates a general failure of control in the developmental process. Many defects, such as hypospadias, imperforate anus, and cleft palate and harelip, are due to a failure to complete the normal process, which a lack of nourishment may to some extent account for. This was almost proved to be the cause of the lion cubs in the Zoological Gardens being born with cleft palates, for with a more liberal and appropriate diet cubs were born normal. But what it is very important to bear in mind is that a defect in development once established is very difficult to eradicate from succeeding generations. What may be called the *tyranny of the ancestor* then comes into operation, in spite of every desire to escape from his vicious and invisible influence. Happily the other side of the problem seems equally true —that a vigorous and manly constitution, with high intellectual qualities, may be transmitted if the parents do not transgress the laws of mental, moral, and physical hygiene. Physical deterioration, with which mental degeneration almost certainly becomes associated, rests on two factors, the defective health of the parents, and the unhealthy surroundings and deficient food of the offspring. Of these two factors the parents exert the greater influence, because in them is summed up not only the result of their own unhealthy lives, but that of their ancestors. Much, no doubt, can be done for the child by good food, good air, and mental and moral training, but he must carry the

tyranny of his type to the end of his days. The Shetland pony, stunted by centuries of bad feeding, cannot in a generation be converted into either a racehorse for speed or into a shire horse for draught. The regeneration will be gradual, however desirous we may be to hasten it. But to the two ends indicated—to inculcate the laws of health among parents, and to secure good food, air, and mental and physical training for the child—to these ends philanthropy may well be directed.

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